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OSTEOMYELITIS OF THE LOWER JAW AND THE RATIONAL USE OF CHEMOTHERAPEUTIC AGENTS

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THE therapeutic aspects of osteomyelitis of the lower jaw can be approached only by a consideration of the pathologic changes of the disease in its relationship to the etiology and anatomic characteristics.

From the standpoint of etiology, it is generally agreed that Staphylococcus aureus is found in about 90 per cent of the cases of osteomyelitis in general. The next commonest organism is Streptococcus. In a certain percentage of cases, there is a mixture of the two types of organisms. Pneumococcus is the third most frequent organism. The identification of the infecting organism has some bearing on the chemotherapeutic aspects of osteomyelitis in relation to the associated bacteremia, which is present in some degree in practically all cases.

The pathologic changes of osteomyelitis of the mandible in relation to its anatomic characteristics are divided into two phases: first, the phase which involves the dissemination of bacteria after they have entered the blood or lymph stream and, second, the phase which involves the fixation of bacteria in tissue and the surrounding of the area with a protecting wall. In retrospect, in the presence of osteomyelitis of the mandible, there is practically always some phase of dentistry. On this point, I quote Thoma's textbook [1] on "Oral Pathology":

> Odontogenic infection is by far the most common cause of suppurative osteomyelitis. The exciting cause is generally the breaking down of the local resistance which, first of all, is caused by disorganization of the protecting pyogenic wall limiting such infection. This may be brought about by injudicious curetting of suppurative lesions containing highly pyogenic organisms; by operative trauma allowing the infection to enter a formerly uninfected area.

It is obvious that bacteria entering the blood or lymph stream from any source may become the etiologic factor in osteomyelitis of the mandible.

Coming now to a consideration of the manner of dissemination of bacteria in the establishment of secondary foci, one assumes that after the bacteria have entered the circulation of the blood and lymph immediately surrounding the area from which they had their origin their move-

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Read before the Southern Section of the American Laryngological, Rhinological and Otological Society, Inc., Charlotte, N. C., Jan. 15, 1945.

1. Thoma, K. H.: Oral Pathology, St. Louis, C. V. Mosby Company, 1941.

ment is determined by the flow of the blood and lymph to the extent that their rate of movement is controlled by the physical laws applying to a solid body placed in a medium of moving liquid. In the areas where the blood and lymph streams are either accelerated or retarded, the movement of the bacteria is altered likewise. Speaking on this point, Forbus [2] stated:

> Typical osteomyelitis begins by the settlement of bacteria in the capillaries of the metaphyseal portion of the bone. The character of the circulation in this region is favorable to bacterial localization since the capillary blood supply is extraordinarily rich and the movement of blood relatively slow because of the large area of the capillary bed. The special significance of the anatomical and physiological condition in this region for the development of pathological processes is emphasized by the frequency with which lesions develop at the line of ossification in a number of other diseases, for example, rickets, scurvy and syphilis.

To these examples of pathologic processes developing in relationship to factors slowing down the infected stream of blood or lymph attention is also called to the bulb of the jugular vein, where thrombosis of the lateral sinus develops in more than 75 per cent of the cases, and to the sublingual area immediately anterior to the hyoid bone, where all cellulitis of the floor of the mouth has its first localization; thrombosis of the cavernous sinus furnishes an example.

May I suggest that osteomyelitis of the frontal bone may be another example of this mechanism of inflammatory reaction? Furstenberg,[3] in his paper on osteomyelitis of the skull, published in 1931, referring to the significance of the suture lines of the skull in the spread of infection, made this observation:

> Histologically, a serrated suture consists of a zone of highly vascular connective tissue. . . . It is a clearly differentiated tissue and by reason of its microscopic structure may possibly function as a barrier to the diffusion of an osteomyelitic process to adjacent cranial bones. . . . If this observation has been correctly made, it suggests, perhaps, a point of value in surgical technic, namely, to limit the removal of bone in operations upon these processes to a suture line whenever it is practicable.

It is obvious from these observations that the areas which are conspicuous for the arrangement of their blood and lymph systems in such a manner as to slow down the streams of blood or lymph are also conspicuous for the fact that they are areas associated with the localization of inflammatory processes caused by infection. The inescapable conclusion is that the localization of inflammatory processes is conditioned on the slowing down of the streams of blood or lymph locally. Except in areas where there has been mechanical traumatism, it is not the rule to observe the initiation of inflammatory processes, except in local areas such as

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2. Forbus, W. D.: Reaction to Injury, Baltimore, Williams & Wilkins Company, 1943.

3. Furstenberg, A. C.: Osteomyelitis of the Skull: The Osteogenetic Processes in the Repair of Cranial Defects, Ann. Otol., Rhin. & Laryng. **40**:996-1012 (Dec.) 1931.

have been related. This would perhaps also apply to the cutaneous tissue if its circulation were fully understood. Now these rules of the dissemination of bacteria may be applied to the anatomic aspects as regards the lower jaw. From the standpoint under consideration, the mandible presents two distinct pictures. In the first place, near the symphysis of the bone there is an extraordinary arrangement of the blood and lymph capillaries similar to the arrangement of the capillaries in the epiphysial area of the long bones, as described by Forbus. Clinical osteomyelitis originating in this space has been observed, as the following report of a case will illustrate.

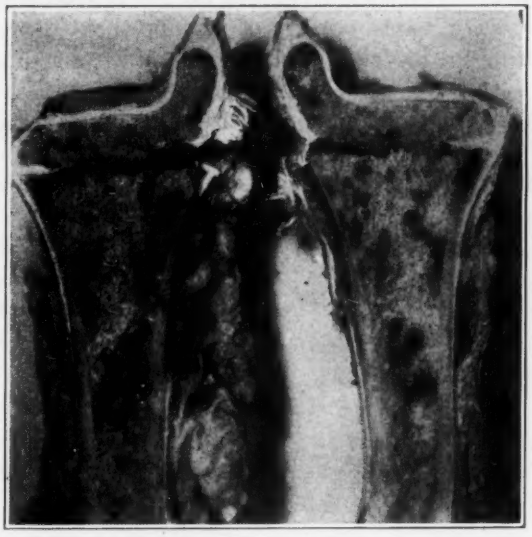

Fig. 1.—"Suppurative Inflammation of Bone: Acute Osteomyelitis of the Lower End of the Tibia.—This bone, which has been split longitudinally, shows the typical development of acute osteomyelitis. The primary lesion is a suppurative inflammatory process in the medullary cavity of the metaphysis; this is represented by the hemorrhagic areas. The second step is the extension of the process to the epiphyseal line across which it extends to reach the periosteum; the destruction of the tissues along this line is shown clearly by the epiphyseal separation. The suppuration next extends beneath the periosteum of the shaft toward the diaphysis; this is shown at the right of the metaphysis of the left half of the split bone" (Forbus, Wiley D.: Reaction to Injury, Baltimore, Williams & Wilkins Company, 1943).

REPORT OF A CASE

A man aged 30 years gave a history of extraction of a lower incisor one week before I saw him. Pain and swelling developed over the chin and corresponding dental area and increased. On examination, there was pronounced boardlike swelling under the chin, extreme swelling of the outer gums and some swelling under the tongue. His temperature was 103 F. Hot magnesium sulfate packs were applied for twenty-four hours, and he grew worse. An incision was made into the deep cellular tissue under the chin, and a large quantity of pus was evacuated. Subsequently, extreme swelling appeared along the lower gums,

spreading laterally as far as the second molar tooth of each side. From time to time, incisions were made over pointing areas, through the periosteum. The areas showed much accumulation of pus in pockets, under which sequestration of bone occurred. The incisions were kept open until the sequestrations were eliminated. After three months, all areas had become dry except the fistulous tract under the chin. With the patient under general anesthesia, a large incision was made and a sequestrum ¾ inch (2 cm.) by ½ inch (1.2 cm.) was removed from the lower border of the bone. Complete recovery followed this promptly (fig. 2).

On the other hand, the portion of the bone posterior to the lower canine tooth has the structural characteristics of an ordinary long bone, but its nutrient blood supply is from both the inside and the outside of the cortex. This is due to the passage of its supplying artery, the inferior alveolar, entering the medullary canal by way of the mandibular foramen and passing out of the canal, after giving off tributaries, through the mental foramen to furnish a supply of blood to the external periosteum

Fig. 2—Large sequestrum and rarefied area in the mandible adjacent to the symphysis.

as well. The definite arrangement of the blood and lymph capillaries associated with the mandible is not known. It can only be surmised. Starr, quoting Lexer, in his memorable monograph on "Acute Hematogenous Osteomyelitis," published in 1922, stated that "The cortex of the entire shaft [of long bone] receives its nutrition from the periosteal vessels."[4]

It seems to me that there is convincing clinical evidence to support the assumption that the blood and lymph capillaries associated with the portion of the mandible posterior to the lower canine offer no obstacle to the free passage of bacteria except in the subperiosteal space. This applies to bacteria coming directly from the infection of the dental field and passing uninterruptedly across the intervening medullary space

4. Starr, C. L.: Acute Hematogenous Osteomyelitis, Arch. Surg. **4**:567-587 (May) 1922.

and the adjacent cortex to the periosteum or through the blood supply of the periosteum, approaching from the outside. This thesis is verified by the clinical finding that a subperiosteal abscess represents the initial finding in osteomyelitis arising from a dental focus.

Roentgenologic diagnosis of mandibular osteomyelitis cannot be made with certainty under two weeks.[5] Interpreted pathologically, retrograde embolism by the bacteria, death of bone by anemia, infarction and formation of a sequestrum by osteoclastic and enzymatic action are necessary before the changes become evident by roentgenologic examination.

Additional evidence for the correctness of this assumption becomes apparent as one proceeds with the discussion of the second phase of the anatomic characteristics of the pathologic changes of this disease, that is, the fixation of bacteria in tissue and surrounding area with a protecting wall. When bacteria gain entrance into the body tissues of a host possessing immunity to those bacteria, the antibodies of the host, under favorable conditions, attack the bacteria by virtue of the affinity existing between the bacteria and the antibodies. Injury results to the tissue cells, and leukotaxine is produced, which causes dilatation of the endothelial walls of adjacent capillaries, thereby increasing their permeability. The dilated capillaries permit the escape of fibrin, which fixes the bacteria in situ by surrounding them with a fibrinous wall. The liberation of the leukotaxine resulting from injury to the tissues causes a rapid immigration of polymorphonuclear leukocytes into the injured tissue, since it also has chemotoxic properties. There is at the same time thrombosis of the blood and lymph capillaries of the involved area. The enveloping wall prevents the escape of bacteria from the inside and at the same time inhibits the passage of substances of whatever character or consistency from the outside into the infected area. The evidences for arriving at these conclusions are summed up by Menkin [6] as follows:

1. Morphologic examination substantiates the view that fixation may well be referable to a lymphatic blockade caused by fibrin and obstructing thrombi in lymphatic channels.

2. It was reasoned that if mechanical obstruction plays an important role in fixation then substances which are unable to disseminate from an acutely inflamed area should for the same reason fail to enter it when injected at the periphery of the area. This is precisely what was found to occur when trypan blue was injected at the periphery of an inflamed focus. Similar results were obtained when a suspension of Serratia

5. Pancoast, H. K.; Pendergrass, E. P., and Schaeffer, J. P.: Head and Neck in Roentgen Diagnosis, Springfield, Ill., Charles C Thomas, Publisher, 1940.

6. Menkin, V.: Dynamics of Inflammation, New York, The Macmillan Company, 1940, pp. 169–174.

marcescens in isotonic solution of sodium chloride was injected into the periphery of a cutaneous inflammatory focus induced by aleuronate (dried and powdered gluten). Burrows has confirmed this experiment with india ink. Studying the living frog under the microscope, Menkin has followed the diffusion of dye from its peripheral inoculation to the boundary of an inflamed area, where its further progress was blocked by the presence of a thrombus occluding a lymphatic channel.

3. Trypan blue injected at the periphery of an inflamed area of the skin injected with a concentrated solution of urea and bacteria (Staph. aureus) penetrates readily into the area whereas it fails to do so when introduced around an inflamed area consequent to injection of distilled water and bacteria (Staph. aureus).[6]

After the inflammatory process is well under way, alteration in the blood supply within the involved tissues causes a disturbance in the intermediary carbohydrate metabolism and glycogen is broken down into lactic acid, favoring acidosis and, consequently, a depletion in the alkali reserve and resulting ultimately in depletion of the alkali reserve and an associated breaking down of the tissue, with the formation of necrotic material and pus.

The foregoing description of an inflammatory process represents the response that all tissue exhibits when invading bacteria are attacked by the tissue antibodies of the host. An inflammatory reaction to bacteria is a rather formal affair. Every step is predicated on some preceding condition. 1. There must be an immunized host possessing antibodies in the tissues. 2. There must be pyogenic bacteria introduced into the blood or lymph stream of the host. 3. The antibodies attack the bacteria, as a result of which leukotaxine is produced, which causes an increase in the permeability of the adjacent capillaries. 4. As a result of the increased permeability, fibrin escapes from the capillaries and fixes the bacteria by forming a fibrinous wall around them. 5. Under the influence of leukotaxine, there is an active migration of leukocytes to the area of inflammation, making a thicker and more substantial wall around the bacteria. 6. There is a retrograde thrombosis of the blood and lymph vessels involved in the inflammatory area, blocking any interchange of fluid or particulate matter from the inside out and vice versa.

This concept of the mechanism of a local inflammatory reaction to infection makes it possible to put on a rational basis the employment of chemotherapeutic agents in the treatment of infectious diseases. It is generally admitted that chemotherapeutic agents such as the sulfonamide drugs and penicillin possess bacteriostatic and bactericidal properties only when they come in direct contact with bacteria. Exclusion of these agents by the protecting mechanical wall from the locally infected area raises a question of doubt as to the value of the chemotherapeutic drugs in purely local infections after they are well established.

It is my practice to give chemotherapeutic drugs as early as possible in cases in which it is suspected that osteomyelitis may develop. It is my belief that it is only in the invasive period of the local situation that the chemotherapeutic agents will have any effect. In the presence of osteomyelitis that has already developed, the chemotherapeutic drugs are given with the hope not that they will influence the local lesion but that they may control infection disseminating into the blood stream from the original focus.

It is needless to attempt to reconcile this concept with the many papers and reports of cases in which striking recoveries from local infections have followed the use of chemotherapeutic agents. In all such cases that have come under my observation, the improvement of the general condition of the patient could be attributed to the action of the drugs on the bacteria in the blood stream, but without any exception the improvement in the local infection has followed localization and drainage of the infected field.

Infections involving the lower jaw offer no exceptions in the general line of treatment of local infections. The chemotherapeutic drugs should be given in every case of this disease to control any tendency toward general infection in the blood stream or metastasis.

Infection of the mandible and its periosteum must be treated specifically, independent of chemotherapeutic agents. During the first twenty-four to forty-eight hours of the infection involving the mandible and its periosteum, I prefer to use ice packs. If this fails to check the progress of the infection, I switch to hot magnesium sulfate packs. Frequent examinations are made for the first appearance of local formation of subperiosteal abscesses and these are incised immediately after their appearance. A small cotton wound applicator used as a probe is helpful in the location of pockets of pus. These pockets are detected by evidences of fluctuation, as shown by palpation with the applicator over the surface of the areas, and also suspected sinus tracts having their origin in the pockets of pus may be detected. Gentleness and perseverance in the search for these pockets of pus are often rewarded by success, when less careful measures result in failure.

Subsequent to the incision and the establishment of drainage from these suppurative areas, there is usually a long period in which a varying degree of necrosis of soft tissue and sequestration of adjacent bone occurs. Any new aspects of drainage which may appear from day to day must be dealt with so as to provide adequate and complete drainage of the separate and individual areas. Necrotic tissue and sequestrated bone should be removed as soon as they can be separated without causing undue trauma to the adjacent tissue. Antiseptics of various kinds may be applied in these wounds. I prefer to make free use of hydrogen peroxide as a cleansing agent.

The evolution of well established osteomyelitis of the mandibular wall is characterized by the long period required for cure, usually from two to six months. I do not believe that radial surgical treatment should be employed. External incision is sometimes necessary in the region of the symphysis menti to drain pus and remove sequestrums in osteomyelitis involving the anterior medullary space as previously described. Whenever external incision is employed, it is my practice to insert a small rubber catheter tube for drainage. Packs of any kind are objectional in that they obstruct rather than promote drainage.

MÉNIÈRE'S SYNDROME: THE BASIC FAULT?

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IS THE basic fault in Ménière's syndrome a dysfunction of the capillaries? It is proposed in this paper to examine the evidence which has accumulated in recent years regarding the pathogenesis of this condition, in order to see how far such a proposition can be justified.

I. THE HISTOLOGIC EVIDENCE

Ménière's disease not being of itself lethal, understanding of it has until recently been greatly handicapped for want of postmortem material. But with the advent of operation in treatment, a few cases of postoperative death have occurred and come to autopsy and a few other actual or analogous cases have been found, so that this gap in knowledge is in process of being filled.

In 1938 Hallpike and Cairns [1] made the first report on the histologic appearances of the temporal bones in typical cases of Ménière's syndrome (describing two ears), and since then the ears in several other characteristic cases have been described [2]—ten ears in all. In each of these a virtually identical histologic picture was found, which consisted in gross dilatation of a large part of the endolymphatic system. In addition, six more ears [3] have been described which showed a similar histologic picture, but the clinical history available in these cases was insufficient to establish the diagnosis.

As a result of these reports, it is coming to be accepted that dilatation of the endolymphatic system is the characteristic pathologic feature in cases of Ménière's syndrome. This dilatation of the endolymphatic

1. Hallpike, C. S., and Cairns, H.: Observations on the Pathology of Ménière's Syndrome, Proc. Roy. Soc. Med. **31**:1317 (Sept.) 1938.

2. (*a*) Yamakawa, K.: Ueber die pathologische Veraenderung bei einem Ménière-Kranken, Ztschr. f. Oto-Rhino-Laryng. **44**:181, 1938. (*b*) Hallpike, C. S., and Wright, A. J.: On the Histological Changes in the Temporal Bones of a Case of Ménière's Disease, Proc. Roy. Soc. Med. **32**:1647 (Oct.) 1939. (*c*) Rollin, H.: Zur Kenntnis des Labyrinthhydrops und des durch ihn bedingten Ménière, Hals-, Nasen- u. Ohrenarzt (Teil I) **31**:73 (March) 1940. (*d*) Lindsay, J. R.: Ménière's Disease: Histopathologic Observations, Arch. Otolaryng. **39**:313 (April) 1944. (*e*) Altmann, F., and Fowler, E. P., Jr.: Histological Findings in Ménière's Symptom Complex, Ann. Otol., Rhin. & Laryng. **52**:52 (March) 1943.

3. Lindsay, J. R.: Labyrinthine Dropsy and Ménière's Disease, Arch. Otolaryng. **35**:853 (June) 1942. Rollin.[2c] Altmann and Fowler.[2e]

space can obviously come only in one of three ways, from deficient absorption of endolymph, from overproduction of it or from a combination of the two. Absorption of endolymph has been shown to take place from the saccus endolymphaticus (Guild[4]), while production is assumed to be by diffusion or secretion from the stria vascularis, in the same way that cerebrospinal fluid is produced from the choroid plexus. A full discussion of these two possibilities will be found in a recent paper by Altmann and Fowler,[2e] who reported 3 of the cases mentioned. It is sufficient to say here that in so far as concerns deficient absorption the evidence as to this possibility is equivocal. Though fibrotic changes in the loose connective tissue around the saccus have been observed in the majority of the cases, similar changes have also been seen in the temporal bones of normal subjects (Hallpike and Cairns). It is therefore felt that these changes are not causative, though they may be contributory to the increased quantity of endolymph.

This leaves overproduction from the stria vascularis as the probable causative factor for the dilatation. Now in the cases described the histologic appearances of the vessels of the stria have been normal. This, however, is what would be expected if the present thesis is correct, for increased permeability of the capillaries is a functional disturbance not demonstrable histologically; it can only be inferred from the changes observed in the surrounding tissues. In the conditions under discussion, that change will be an increase in the quantity of endolymph, which has been found in every case examined.

II. THE EVIDENCE OF CLINICAL EXPERIMENT

1. Abnormal Water Metabolism.—Mygind and Dederding[5] have produced evidence to show that in susceptible subjects hyperhydration can bring on attacks and that dehydration can abolish them. They have also offered a wealth of collateral clinical evidence to support their thesis of a fault in water metabolism, such evidence as the frequent occurrence in association with the attacks of vertigo, of headache, vasomotor rhinitis, "rheumatism" and water depots in the subcutaneous tissues. Postulating, before the histologic picture was known, cellular edema due to capillary dysfunction as the explanation of their findings, they instituted treatment by means of limited intake of salt and fluids for relief of the condition and claimed good results by this method. They sought, in effect, to diminish leakage from the reservoir by limiting the water supply.

4. Guild, S. R.: Circulation of Endolymph, Am. J. Anat. **39**:57 (March) 1927.

5. Mygind, S. H., and Dederding, D.: Clinical Experiments with Reference to the Influence of Water Metabolism on the Ear, Ann. Otol., Rhin. & Laryng. **47**:360 (June) 1938.

2. The Sodium Ion Content of the Blood Serum.—Furstenberg and his associates,[6] not satisfied with the explanation of Mygind and Dederding, sought to incriminate the sodium ion. They produced laboratory evidence to support their contention, but this evidence has not stood up to later investigation (Furstenberg[7] and Talbott and Brown).[8] Nevertheless, clinically they claimed good results by dietetic treatment with a salt-free diet low in sodium together with the administration of large doses of ammonium chloride, an acid-producing but also a diuretic drug. This is only a variation on the theme of Mygind and Dederding. They lower the amount of water available, and Furstenberg diminishes the avidity of the surrounding terrain for it. Consciously or not, Furstenberg seemed to accept the postulate of an increased permeability of the capillaries and of a liability to tissue edema in patients with Ménière's disease.

3. The Potassium Ion Content of the Blood Serum.—Talbott and Brown[8] approached the problem from a slightly different angle. They investigated the concentration of electrolytes and the hydration of the blood in patients with Ménière's syndrome and found no consistent changes from the normal. Since, however, the proportion of sodium to potassium in most normal diets is approximately parity, it follows that a diminished intake of sodium involves a relative increase in potassium content. As they had in mind the results of Furstenberg and this fact, it seemed to them logical to try the effect of adding potassium to a normal diet. They substituted potassium chloride for sodium chloride at table and added further considerable quantities; they reported beneficial results in all of 40 cases, though cure in none. However, as Talbott and Brown themselves pointed out, potassium salts have a diuretic action, which hastens the excretion of water and of sodium chloride, and it is possibly to this effect that the improvement was due. If this is the explanation, the exhibition of potassium will, like methods 1 and 2, decrease the leak in the tissues by diminishing the quantity of available fluid, but it does not, any more than they, do anything to repair the walls of the reservoir.

4. Histamine and the Primary Vasodilator Group.—Here one enters even more difficult territory. In 1940 Shelden and Horton[9] reported

6. Furstenberg, A. C.; Lashmet, F. H., and Lathrop, F.: Ménière's Symptom Complex: Medical Treatment, Ann. Otol., Rhin. & Laryng. **43**:1035 (Dec.) 1934.

7. Furstenberg, A. C.; Richardson, G., and Lathrop, F.: Ménière's Disease: Addenda to Medical Therapy, Arch. Otolaryng. **34**:1083 (Dec.) 1941.

8. Talbott, J. H., and Brown, M. R.: Ménière's Syndrome: Acid-Base Constituents of the Blood; Treatment with Potassium Chloride, J. A. M. A. **114**: 125 (Jan. 13) 1940.

(Footnotes continued on next page)

dramatic results in the relief of acute attacks by intravenous administration of histamine. In 1941 Atkinson[10] reported that by means of an intradermal test with histamine it is possible to divide patients with Ménière's syndrome into two groups—those who are unduly sensitive and those who give a normal response.[11]; that only the patients who show an abnormal response, that is to say, those who show primary dysfunction of the capillaries as evidenced by their extreme response to minute quantities of histamine intradermally, are improved by desensitization, and that those who show a normal response are made worse by desensitization. On the face of things, there appears to be a contradiction, since Shelden and Horton obtained their results on all patients indiscriminately. They did not attempt to determine beforehand the sensitivity of the patient to histamine and so may be assumed to have treated some patients of both groups successfully. This contradiction is only apparent, but before the explanation of it is gone into the position as regards Atkinson's second group 1 must be examined.

5. Nicotinic Acid and the Primary Vasoconstrictor Group.—Assuming that an abnormal response to a cutaneous test with histamine indicated a primary vasodilator mechanism, Atkinson postulated that the attacks in patients who gave a normal response might well be due to vasospasm, a primary vasoconstrictor mechanism. He was able to show by clinical experiments that in some instances, at least, this is indeed so, that in patients belonging to this group attacks could be induced with vasoconstrictor drugs and abolished or diminished by vasodilator drugs. He concluded that the postulate of vasospasm in this group was at least in part correct.

Now the effect of vasospasm is to diminish the supply of oxygen to the tissues and, in the first place, to the capillaries, and this partial anoxemia, by interfering with the nutrition of the capillary wall, increases capillary permeability. The ultimate effect, a local tissue edema, is therefore the same as in the previous group, though the

9. Shelden, C. H., and Horton, B. T.: Treatment of Ménière's Disease with Histamine Administered Intravenously, Proc. Staff Meet., Mayo Clin. **15**:17 (Jan. 10) 1940.

10. Atkinson, M.: Observations on the Etiology and Treatment of Ménière's Syndrome, J. A. M. A. **116**:1753 (April 19) 1941.

11. Owing to a misconception both of the method of performing the test and apparently of its implications and limitations, the validity of this test has been called in question by some members of the Mayo group (Williams, L. H.: Intrinsic Allergy as It Affects the Ear, Nose and Throat: The Intrinsic Allergy Syndrome, Ann. Otol., Rhin. & Laryng. **53**:397 [Sept.] 1944. Lillie, H. I.; Horton, B. T., and Thornell, W. C.: Ménière's Symptom Complex: Observations on Hearing of Patients Treated with Histamine, ibid. **53**:717 [Dec.] 1944). A paper on this subject, which it is hoped will clear the air, has recently been published (Atkinson, M.: Ménière's Syndrome: The Validity of the Intradermal Histamine Test, Ann. Otol., Rhin. & Laryng. **54**:801 [Dec.] 1945).

mechanism of production is different. In one group increased capillary permeability is the result of abnormal sensitivity to histamine or a histamine-like substance, and in the other it is the result of anoxemia. The anoxemic effect can be overcome by any means which will increase the vascular supply to the part. Since the vascular supply of the inner ear cannot be affected directly by such physical means as massage and heat or easily by blocking vasoconstrictor impulses (though this can be done indirectly by a block of the stellate ganglion) vasodilator drugs are the method of choice. Atkinson used nicotinic acid, a powerful vasodilator drug, for this purpose and has been able to procure good results by this method.[12]

Histamine.—To return to histamine, how is the apparent contradiction to be explained that histamine administered intravenously in large dosage seems to benefit at least temporarily all patients regardless of their grouping? As far as concerns the capillaries, histamine has a dual action—it increases capillary permeability and it produces vasodilatation. It is therefore suggested that the reason why Shelden and Horton were able to obtain improvement in all cases is that in the histamine-sensitive group they brought about a rapid desensitization and thus decreased the liability of the capillaries to leak under the action of histamine, while in the histamine-insensitive or vasospastic group they overcame the vasospasm through the vasodilator action of the drug. Thus intravenous injections of histamine can be effective in both groups. But if a continuance of its effect is attempted in the vasospastic group by means of repeated subcutaneous injections of a small dose, not only will the attempt fail but it will often produce a resistance to other and more efficacious methods (Atkinson[13]).

One can sum up the position as regards the use of histamine in Ménière's syndrome, therefore, by saying that it affects function of the capillaries in two ways. Given intravenously in a single large dose or subcutaneously in repeated small doses, it can apparently produce an immunity to its own action and thus benefit materially the group which has a primary dysfunction of the capillaries. Given in a single large dose, it will, owing to its vasodilator action, benefit also the group which is due to anoxemia caused by vasospasm, even if only temporarily.

6. Allergy.—A number of cases of typical Ménière's syndrome indisputably due to a protein sensitivity have been reported in the literature.[14] This does not profess to be a comprehensive list, and

12. Atkinson, M.: Ménière's Syndrome: Results of Treatment with Nicotinic Acid in the Vasoconstrictor Group, Arch. Otolaryng. **40**:101 (Aug.) 1944.

13. Atkinson, M.: Histamine in the Treatment of Ménière's Syndrome: An Appraisal, J. A. M. A. **119**:4 (May 2) 1942.

(Footnotes continued on next page)

those given are examples only. Here again, however, is a process which induces the characteristic syndrome and in which the major disturbance of physiologic function is an increased permeability of the capillaries.

7. Avitaminosis-C.—Ohnell [15] was the first to suggest that a deficiency of vitamin C might be a cause of Ménière's syndrome. Adam [16] reported 2 cases, which are not entirely satisfactory. The suggestion is by no means fully substantiated, but it is of interest in the present connection because here again dysfunction of the capillaries comes into the picture.

8. Toxic Labyrinthitis.—Wright [2b] maintained that a low grade focal infection producing toxic labyrinthitis is the usual cause of Ménière attacks. Though this theory is not generally accepted, it certainly has validity in some cases. Now an infective process produces a local capillary response—dilatation and increased permeability—so that this mechanism will also fit into the thesis.

CONCLUSION

When all this evidence is put together, the case for dysfunction of the capillaries as the basic fault which produces the disturbance of labyrinthine function which leads to attacks is a strong one. An undue permeability of the capillaries explains the histologic picture. Infection, allergy and avitaminosis-C are primary disturbances of function of the capillaries. So also is sensitivity to histamine, which is probably related to demonstrable protein sensitivity. Vasospasm produces secondarily the same effect on the capillary walls by interfering with their nutrition. The methods of treatment by reduction of water intake and sodium intake or by increase of potassium intake owe their beneficial effect to diminishing the amount of fluid available to seep through the abnormally permeable capillary walls. Treatment by histamine or nicotinic acid in the appropriate groups is more fundamental, since it seeks to overcome the factor which is damaging the capillary wall. The one tries to limit the leak from the reservoir by diminishing the head of water; the other attempts to repair the walls.

14. Duke, W. W.: Ménière's Syndrome Caused by Allergy, J. A. M. A. **81**: 2179 (Dec. 29) 1923. Dohlman, G.: Menieresche Krankheit auf allergischer Basis, Acta oto-laryng. **27**:245, 1939. Hartsook, N. E.: A General Consideration of Defective Hearing and Deafness, with Particular Reference to Etiology, Ann. Otol., Rhin. & Laryng. **46**:510 (June) 1937. Jones, M. F.: Manifestations of Allergy in the Ear, ibid. **47**:910 (Dec.) 1938.

15. Ohnell, H.: Aktuelle Skorbutfragen, Arch. f. Verdauungskr. **51**:281 (May) 1932.

16. Adam, J.: Ménière's Syndrome and Avitaminosis, J. Laryng. & Otol. **54**: 256 (May) 1939.

If this thesis is accepted, the major difficulty of Ménière's disease *(la maladie de Ménière)* is resolved. It is in actual fact not a disease per se but a syndrome. It is the syndrome of the labyrinth in paroxysmal form and can be produced by any factor which, by increasing the permeability of the capillaries forming the stria vascularis, increases the production of endolymph, thus raising intralabyrinthine pressure. Satisfactory treatment depends, therefore, on determining what that factor is and eliminating it. This is not always easy, but it becomes less difficult when the various possible factors are appreciated.

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WAR INJURIES TO THE MASTOID AND THE FACIAL NERVE

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IN A SERIES of 4,400 battle casualties requiring surgical treatment admitted to an army general hospital overseas, there were 21 patients with injury to the mastoid portion of the temporal bone or to the facial nerve or to both. Of this number there were 6 patients (cases 1, 2, 3, 4, 5 and 6) with injury to the mastoid area. Two patients (cases 7 and 8) sustained injury to the mastoid area with involvement of the facial nerve. Four patients (cases 9, 10, 11 and 12) had injuries to the facial nerve producing paralysis, with no evidence of injury to the mastoid structures.

A perusal of the literature reveals that little has been written concerning this particular type of war injury. Many of these patients are treated in forward areas by the general surgeon, and it was thought to be worth while to point out some of the lessons which have been learned concerning their treatment.

REPORTS OF CASES

CASE 1.—A private first class, aged 22, was wounded in action on Oct. 20, 1944, when he was struck by several shell fragments, one of which produced a severe perforating wound of the right eye. Other fragments struck the left mastoid process. The cortex of the mastoid was fragmented, and a spicule of bone was driven into the external auditory canal, producing a laceration of the membranous canal wall and a rupture of the tympanic membrane. The patient was given sulfadiazine from the moment of injury. At the time of his admission to the hospital, the right ear was suppurating. Roentgen examination was made on October 26 by Major W. A. Evans:

"Left Orbit and Skull: Examination in the standard projections shows no evidence of a foreign body about the left orbit, but there is a minute metallic fragment in the region of the lateral aspect of the right orbit. The cranial vault and the facial bones appear intact, but there is a limited injury to the lateral aspect of the left mastoid area. The sphenoid and petrous ridges are symmetric and intact."

The patient was observed until November 3, during which time the right eye was enucleated and he was treated with sulfadiazine, penicillin and three transfusions of whole blood. On this date mastoidectomy was performed on the left side. The anesthetic was pentothal sodium given intravenously with oxygen administered through an intratracheal tube. At operation a large sequestrum was found which involved the mastoid cortex, the mastoid cavity and the posterior osseous canal wall. The entire mastoid cavity was full of pus, blood clots, granulation tissue and fragmented bone. A complete mastoidectomy was done. A 15 F

rubber catheter was introduced into the mastoid cavity, and the posterior edge of the mastoid incision was sutured to it. The external auditory canal was firmly packed with petrolatum gauze. Postoperatively, 5 cc. of a solution of penicillin containing 1,500 units per cubic centimeter was introduced through the catheter four times daily. This procedure was continued for five days after the operation. There was then no further suppuration, and the tube was removed. In addition penicillin was given intramuscularly, 25,000 units every three hours, day and night.

The patient made an uneventful recovery. The laceration of the external auditory canal and the perforation of the tympanic membrane healed completely.

CASE 2.—A sergeant, aged 24, was wounded in action on Nov. 14, 1944, at which time a metallic fragment 1 cm. in diameter entered the left squamous portion of the temporal bone, just anterior to the helix. On November 15 (at a

Fig. 1 (case 2).—Large defect in the left inferior parietotemporal area, at the point of entrance of the missile.

hospital in the forward area) a débridement of the skull wound was done with removal of the devitalized brain tissue and the foreign body. Roentgen examination revealed that a second foreign body was present in the mastoid portion of the temporal bone.

The patient was evacuated to this hospital. At the initial examination a hernia cerebri was present in the left temporal region. There was a profuse purulent discharge from the left external auditory canal, and a large perforation was present in the left tympanic membrane. There was three point tenderness over the mastoid. The neurologic examination was negative in result. The ocular fundi were normal. A roentgen examination of the temporal bone on November 28 by Major W. A. Evans revealed the following:

"Skull: Examination in the four standard projections together with an exposure in the chin-nose position and exposures in Law's and Stenver's projections to

show the mastoid areas reveals a defect 4 by 2 cm. in the left inferior parietal-temporal area, and there is a linear fracture about 7.5 cm. in length in the left parietal bone (fig. 1). Other linear fractures extend anteriorly from the defect through the lower frontal bone into the frontal sinus. Two small detached fragments of bone are seen in the anterior aspect of the defect. Two minute metallic fragments are seen in the soft tissues in the scalp at the anterior end of the defect and two other minute metallic fragments are seen in the substance of the frontal lobe. A larger metallic fragment 0.6 by 0.9 cm. (fig. 2) is seen in the left mastoid area, where there has been fragmentation of some of the trabeculae near the arcuate eminence. The mastoid cells appear pneumatic, however, and the facial bones appear intact with the paranasal sinuses pneumatic on both sides. Normal relations are maintained at the temporomandibular joints, and the sella turcica appears normal."

The patient's general condition was rather poor at this time. He was given a course of sulfadiazine orally and penicillin, 25,000 units administered intramuscularly every four hours day and night, three transfusions of whole blood, forced feeding of a high caloric diet and multivitamin therapy.

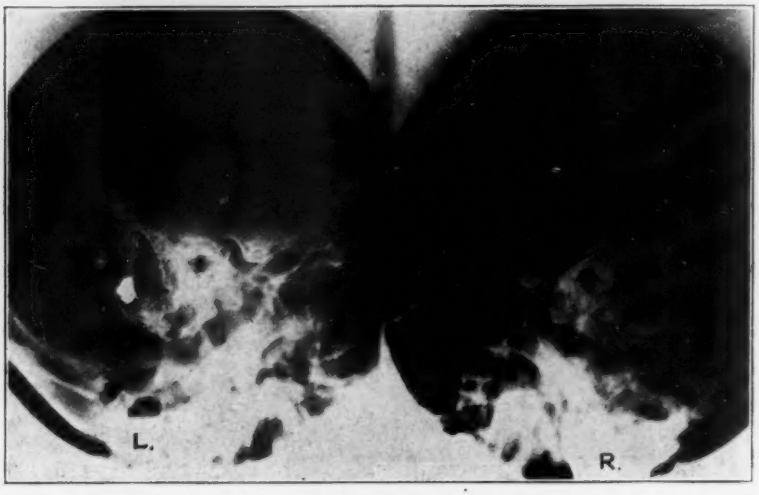

Fig. 2 (case 2).—Large metallic foreign body in the left mastoid area, in the region of the posterior border of the lateral sinus.

On December 11 mastoidectomy was done on the left side with the patient under anesthesia with pentothal sodium given intravenously and oxygen, through an intratracheal tube. A postauricular incision was made, and the mastoid cells were found full of a peculiar green-blue fluid. The mucous membrane lining the mastoid cells had the same peculiar color and was much thickened and degenerated. The mastoid tip was fractured. The dural plate of the mastoid antrum and of the middle ear was fractured and depressed over the temporal lobe. This was elevated and removed. The mastoid portion of the lateral sinus was completely uncovered, and the metallic foreign body, approximately 0.5 by 0.3 cm., was found to have perforated the posterior wall of the lateral sinus. There was no bleeding following its removal, and the sinus filled normally when pressure was made on the left internal jugular vein. The mastoid incision was extended about 3.0 cm. to the temporal parietal region. A mass of granulation tissue, skin and scar tissue

was removed. The operative defect in the temporal parietal region was inspected. Bone edges were freshened. A small rubber tube was placed in the mastoid antrum and sutured to the edge of the mastoid wound. The cavity was dusted with sulfanilamide powder and the wound closed.

Postoperatively the intramuscular administration of penicillin (25,000 units every three hours day and night) was continued for five days. In addition, through the tube in the postauricular wound a solution of penicillin, 1,500 units per cubic centimeter, was introduced every two hours during the day and every four hours during the night. The tube was removed on the sixth postoperative day. The wounds healed by first intention. The patient's course was uneventful, and recovery was complete without residuals.

CASE 3.—A private, aged 24, was wounded in action on March 15, 1945, when a .45 caliber bullet struck the left mastoid, perforating the cortex of the mastoid and the posterior and the anterior auditory canal, and lodged in the soft tissues of the cheek anterior to the ramus of the mandible. On March 16 at a forward hospital an operation was done which consisted of removal of the foreign body and a partial debridement of the left mastoid. The patient was evacuated and admitted to this hospital on March 30. Examination at that time revealed a profuse purulent discharge from the right external auditory canal. There was a great amount of dense scar tissue which almost completely obliterated the external auditory canal, and spicules of bone from the fragmented posterior canal could be seen protruding into the external auditory canal. There was a partially healed postauricular wound from which a great amount of pus was draining. Roentgen examination by Major W. A. Evans on the same date revealed the following:

"Mastoid Areas: Examination in Law's and Stenver's projections shows an essentially normal appearance of the structure on the right. A small operative defect is seen on the left, but a large part of the cellular structure remains, and the cells appear infiltrated with the trabeculae undergoing dissolution."

The patient had been receiving sulfadiazine orally and penicillin intramuscularly since the time of injury.

In view of the foregoing findings, on April 9 a radical mastoidectomy was done with the patient under anesthesia with pentothal sodium given intravenously and oxygen given through an intratracheal tube. The usual postauricular mastoid incision was made. The periosteum was elevated. A small operative defect was noted at the site of the previous débridement. This cavity was full of scar tissue and purulent exudate. There was a considerable comminution of the bone of the mastoid and of the posterior osseous wall of the external auditory canal. The structures of the middle ear had been destroyed. The simple mastoidectomy was completed, a depressed fracture of the anterior wall of the lateral sinus was found, and this was elevated and removed. When the lateral sinus was uncovered it was found to be thrombosed. The vein was bluish white in appearance and firm to touch. The sinus did not fill when pressure was made on the internal jugular vein. The lateral sinus was incised, a firm thrombus was noted and there was no purulent drainage. The sinus was not further disturbed, and no attempt was made to remove the thrombus. The radical mastoidectomy was then completed.

The patient's postoperative course was uneventful. Administration of penicillin, 25,000 units every four hours day and night, was continued until the fifth postoperative day. A small rubber tube had been left in the mastoid cavity, and penicillin solution, 1,500 units per cubic centimeter, was introduced every two

hours during the day. The mastoid wound was healing satisfactorily when the patient was evacuated to the zone of the interior.

CASE 4.—A private first class, aged 21, was wounded in action on Feb. 17, 1945, when a large shell fragment entered the left occipital portion of the skull, just posterior to the mastoid articulation. It then passed through the mastoid process and the posterior bony canal wall destroying the tympanic membrane and the structures of the middle ear, then through the anterior canal wall and finally lodged in the region of the left temporomandibular joint. On February 17 an operation was done at a hospital in the forward area, at which time the fragment was removed through the same path through which it had entered. At that time no attempt was made to inspect or to debride the structures of the mastoid or the middle ear. The patient was kept under treatment with sulfadiazine orally and intramuscular injections of penicillin, and was evacuated to a general hospital, arriving March 24.

At the initial examination a draining fistula was present at the site of the wound entrance. There was a great amount of purulent discharge in the left external auditory canal. The tympanic membrane and the ossicles had been destroyed so that the promontory of the middle ear was easily visible after the ear canal had been cleaned. It was possible to pass a probe through the opening in the posterior canal wall directly into the cavity of the mastoid. Roentgen examination, made on March 25 by Major W. A. Evans, revealed the following:

"Mastoid Areas: Examination in the Law and the Stenver projection shows a limited cellular structure on both sides, and there is some periantral sclerosis, indicating an old bilateral inflammatory residue. There has been an extensive comminution of the left mastoid process with a wide scattering of the smaller bone fragments. No foreign body is seen, and the petrous ridges appear intact."

In view of the persistent draining fistula and other clinical and roentgenologic findings, it was decided that a radical mastoidectomy was indicated. This was done on March 31 with the patient under anesthesia with sodium pentothal and with oxygen given through an intratracheal tube.

The usual posterior auricular incision was made. On elevating the periosteum there was revealed a fracture which extended through the tip of the mastoid process and the posterior bony canal wall. The fractured portion of the mastoid was carefully elevated and dissected free. The mastoid cells were found to be full of necrotic bone, pus and organized blood clot. The remaining portion of the posterior bony canal was removed, and the middle ear and mastoid cavity were converted into one. The cavity was dusted with sulfonamide crystals. A petrolatum gauze pack was inserted and a closure effected.

Postoperatively, the patient was given penicillin intramuscularly, 25,000 units every four hours. This was continued for one week. The healing was satisfactory, and the patient was evacuated to the zone of the interior with the wound in good condition.

CASE 5.—A corporal, aged 23, was wounded in action on Jan. 17, 1945, while in combat on Luzon, Philippine Islands. Multiple shell fragments struck the patient, producing numerous wounds over the body. Two large metallic fragments struck the left auricle. The wound of entrance was through the concha of the left ear. The fragments then perforated the posterior osseous canal wall and entered the left mastoid cavity. The patient received immediate care in the forward area, and sulfadiazine therapy was started orally and penicillin intramuscularly. He was received at this hospital on February 3. The initial examination revealed that, in addition to his other wounds (which are not dis-

cussed in this presentation), he presented a large wound of entrance in the left concha. There was no wound of exit. There was a great deal of purulent drainage from the left external auditory canal and from the wound of entrance. There was postauricular edema and three point mastoid tenderness on palpation. The patient's general condition was good. A roentgen examination was made on February 3. The report by Major W. A. Evans is as follows:

"Mastoid Areas: Examination in the Law and the Stenver projection shows two metallic fragments in the mastoid area on the left, the larger of these measuring 1.1 by 0.8 cm. It has penetrated the cellular structure to a depth of about 2.5 cm. just posterior to the canal, and there is an irregular defect in the cells representing the path of the foreign body. There is some clouding of the cells on the left, particularly about the foreign body, as compared with the right, but there is no evidence of any bone destruction except that incident to the passage of the foreign body. The cellular development is moderate and symmetric. The cranial vault is of normal thickness and structure, and no fracture or separation at the suture lines is demonstrable. There are no roentgen signs of increased intracranial pressure or of abnormal intracranial calcification. The sella turcica appears normal, and the sphenoid and petrous ridges appear intact."

On February 5 mastoidectomy was performed on the left side. The foreign bodies were in the cavity of the mastoid. At the operation it was noted that the foreign bodies had produced a great deal of destruction. There was considerable pus present in the mastoid cavity, with thickening of the mucous membrane, granulation tissue and necrotic and splintered bone. A complete mastoidectomy was done.

The postoperative course was uneventful. The mastoid cavity was treated by instilling through a small rubber catheter 5 cc. of a solution of penicillin containing 1,500 units per cubic centimeter every two hours during the day. The tube was removed on the fifth postoperative day. The wounds healed by first intention, and the middle ear was dry at the time he was evacuated.

CASE 6.—A sergeant, aged 33, was wounded in action on Jan. 20, 1945, on Luzon, Philippine Islands. A large metallic foreign body made its entrance in the region of the left temporomandibular joint just anterior to the tragus. It then passed through the external auditory canal and entered the lower third of the left mastoid process. First aid treatment was given, and the patient was hospitalized. On February 1 at a hospital in the forward area an operation was done for removal of the foreign body in the mastoid. A small incision was made over the tip of the mastoid process and with a hemostat the foreign body was grasped and removed. A drain was inserted into the mastoid cavity. The patient arrived at this hospital on February 3. At the initial examination the drain was removed from the mastoid wound, and considerable discharge from the mastoid cavity and middle ear was seen. The roentgen report of February 5 by Major W. A. Evans is as follows:

"Mastoid Areas: Examination in the Law and the Stenver projection shows an entirely normal appearance of the structure on the right. There is clouding throughout the structure on the left, apparently due in part to overlying soft tissue swelling and in part to inflammation of the cells. There is comminution of the cells at the lateral aspect of the left mastoid process. No foreign body is demonstrable, and the petrous ridges appear intact."

On February 14 complete mastoidectomy was done on the left side with the patient under anesthesia by the intravenous injection of a 2.5 per cent solution of pentothal sodium and intratracheal administration of oxygen. At this time a large

cavity was found in the left mastoid process. There was a great amount of destruction present in the mastoid cells, with considerable granulation tissue and necrotic bone. At the completion of the operation a small rubber catheter was left in the mastoid cavity.

The postoperative course was uneventful. The mastoid wound was treated by instilling through the catheter 5 cc. of a solution of penicillin containing 1,500 units per cubic centimeter every two hours during the day. The tube was removed on the fifth day. The wound closed rapidly after the tube was removed. The middle ear was dry at the end of ten days.

CASE 7.—A private, aged 22, was wounded Oct. 29, 1944, on Leyte, Philippine Islands. At that time a large metallic fragment, approximately 1.3 by 1.3 cm., entered the region of the tip of the right mastoid process of the temporal bone (fig. 3 *A*). There was no wound of exit. He received emergency treatment in the forward area and was admitted to this hospital on November 10. Examination on the day of admission revealed that the patient's general condition was good. There was profuse purulent discharge from the right external auditory

Fig. 3 (case 7).—*A*, the arrow points to the healed scar at the point of entrance of the foreign body in the right mastoid area. *B*, complete peripheral paralysis of the right facial nerve as a result of the injury described in the text.

canal and complete right peripheral facial paralysis (fig. 3 *B*). Roentgenologic examination on November 11 by Major W. A. Evans revealed the following:

"Skull: Examination in the four standard projections shows a metallic fragment 1.3 by 1.3 cm. in the neck behind the right ascending ramus of the mandible, close to the base of the skull and the tip of the mastoid process. A minute metallic fragment is also seen in this area (fig. 4). The cranial vault is of normal thickness and structure, and no fracture is demonstrable. The sella turcica appears normal, and the sphenoid and petrous ridges are symmetric and intact. The mastoid structures and the paranasal sinuses are pneumatic."

On November 15 an operation was done on the right mastoid and the right facial nerve. The usual postauricular mastoid incision was made, which was extended anteriorly to the angle of the jaw. The mastoid cortex was exposed, and a complete mastoidectomy was performed. During the surgical treatment it was found that the large metallic fragment had struck the tip of the mastoid a glancing blow, producing a fracture of the mastoid portion of the temporal

bone and involving the facial nerve. The foreign body was found to be lodged in the region of the styloid process. The bone of the mastoid process was necrotic, and there was a great amount of granulation tissue and polypoid degeneration of the mucosa. The facial canal was completely uncapped, and it was found that the nerve had been torn at a point 2 mm. below the external semicircular canal. The dissection was continued anteriorly into the substance of the parotid gland. The large metallic foreign body was located and removed. The injury to the facial nerve extended to a point just posterior to the division of the nerve in the parotid gland. The cranial and peripheral ends of the facial nerve were located, the enveloping scar tissue was removed and the nerve ends freshened. A nerve graft approximately 2 inches (5 cm.) long was taken from the left anterior femoral cutaneous nerve after the technic described by Ballance and Duell. This graft was sutured in place with two strands of no. 8.0 arterial silk passed through the nerve sheath. At the point of suture the

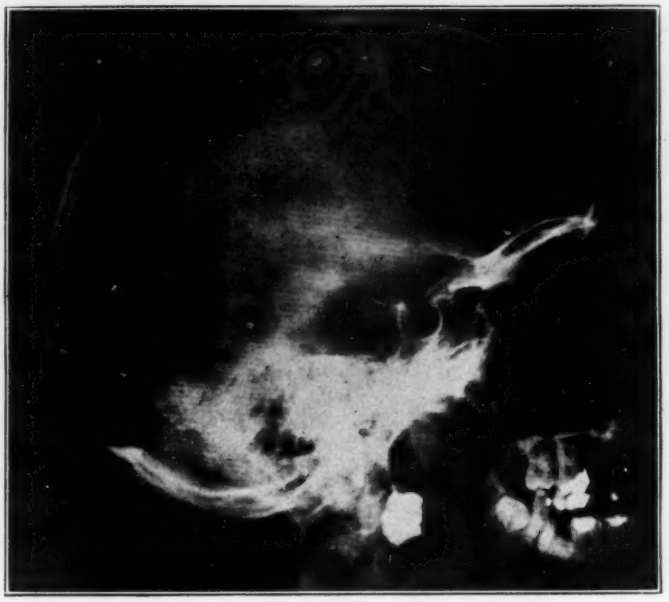

Fig. 4 (case 7).—Lateral view of the skull illustrating the location of the large foreign body.

nerve was covered with small strips of fascia lata. The wound was closed, and a rubber catheter was introduced into the wound through which 5 cc. of solution of penicillin (1,500 units per cubic centimeter) was introduced every two hours during the day and every four hours at night.

The postoperative course was uneventful. The mastoid wound healed by first intention. During the postoperative period the patient was given daily physical therapy and instructed in massage and exercise of the facial muscles. A splint of acryllic plastic was made which supported the corner of the mouth and the lower facial muscles. The patient was evacuated to the zone of the interior, where the physical therapeutic measures were continued. In a personal letter recently received, the patient reported that return of function to the right side of the face had occurred and that he was able at this time to close the right eye

and move the muscle on the right side of the face. Recovery had taken place approximately seven months after the nerve graft operation.

CASE 8.—A technical sergeant, aged 29, was wounded in action on Nov. 6, 1944, on Leyte, Philippine Islands, when a large shell fragment struck the left side of his skull in the region of the left external auditory canal. First aid treatment was quickly given, and the patient was taken to an evacuation hospital where he was kept for several weeks. A débridement of the wound was done, and penicillin and sulfadiazine therapy was instituted. When his condition warranted it, he was evacuated to this hospital and was admitted on December 10.

A physical examination made on admission revealed the patient's general condition to be excellent. There was a large wound of entry (fig. 5 *A*) involving the tragus and the concha of the left ear. There was loss of the external auditory canal, the anterior third of the mastoid and the structures of the middle ear. There was a great amount of purulent drainage present in the wound. Numerous spicules of bone were seen. The patient had complete peripheral paralysis

Fig. 5 (case 8).—*A*, the wound of entrance, as indicated by the arrow. The missile destroyed the tragus and entered the external auditory canal. *B*, complete peripheral facial paralysis sustained by the patient as the result of the injury.

of the left facial nerve (fig. 5 *B*). Roentgen examination by Major W. A. Evans made on December 11 and 13 revealed the following:

"Skull: Examination in the four standard projections shows scattered small metallic fragments in the region of the left temporomandibular joint, well outside the cranial cavity. The cranial vault is of normal thickness and structure with no fracture demonstrable. The pineal gland contains a calcium deposit and is seen to occupy a normal midline position. The sella turcica appears normal, and the sphenoid and petrous ridges are symmetric and intact. The paranasal sinuses are pneumatic on both sides, but there is some clouding of the mastoid cells on the left, and there is, we think, a fracture through the tip of the mastoid process.

"Mastoid Areas: Examination in the Law and the Stenver projections shows a large defect in the region of the external auditory canal on the left side with extensive comminution of the mastoid process, and the fracture lines enter the petrous ridge just lateral to the arcuate eminence. Several metallic fragments

are seen in the left mastoid area. The mastoid structures on the right appear clear."

The patient was prepared for operation with the use of penicillin intramuscularly. This was given in dosages of 25,000 units every three hours day and night. The wound was treated locally with continuous soaks of penicillin solution (1,500 units per cubic centimeter).

On November 20 the following surgical treatment was given. A postauricular incision was made, the periosteum of the mastoid was elevated, and the mastoid cavity was exposed. The mastoid cortex of the remaining portion of the mastoid process was removed. The necrotic cells, fragmented bone, pus and granulation tissue and metallic fragments were removed. During the course of the exploration a depressed fracture of the tegmen of the tympanum and the antrum was found. The depressed bone was elevated and removed. The dura was discolored, and some granulations were present over this area. There was also a depressed fracture of the lateral sinus plate. This was elevated and removed. The lateral sinus was covered with granulation tissue. The sinus was found to fill normally when pressure was made over the left internal jugular vein and therefore was not further disturbed. The facial canal was explored in its entirety. A fracture of the facial canal was found which impinged on the nerve at a point 3 mm. below the external semicircular canal. The fractured bone was removed and the entire facial nerve uncovered to its point of exit at the styloid mastoid foramen. At the point of the fracture a hemorrhagic area was found in the nerve several millimeters in length. An external and internal neurolysis of the facial nerve was done. The exposed facial nerve was covered with a piece of fascia from the temporalis muscle. The mastoid wound was closed and a rubber tube inserted into the mastoid cavity. Through this tube a solution of penicillin (1,500 units per cubic centimeter) was introduced every four hours. The postoperative course was uneventful. Physical therapy was given on the fifth postoperative day. The patient was evacuated to the zone of the interior for follow-up care.

CASE 9.—A private first class, aged 21, was wounded in action on Oct. 31, 1944. At this time he received a gunshot wound. The caliber of the missile is unknown. The wound of entrance was in the left cheek at a point inferior to the malar eminence. The point of exit was just below the tip of the right mastoid process. First aid treatment was given in the forward area, and the patient was evacuated to this hospital, where he arrived on November 12.

At the initial examination the wounds of entrance and exit were well healed (fig. 6). There was a complete peripheral paralysis of the right facial nerve. The patient's general physical condition was good. Electrical tests for the function of the seventh nerve were done which revealed a loss of response to the faradic current and good response to the galvanic current. The roentgen studies on November 14 by Major W. A. Evans revealed the following:

"Skull: Examination in the four standard projections together with a study in the chin-nose position shows a cranial vault of normal thickness and structure, and no fracture or foreign body is demonstrable. The facial bones also appear intact, and the paranasal sinuses are pneumatic on both sides. There is some thickening of the mucosa in the left antrum, but this has the appearance of an old inflammatory residue. Normal relations are maintained at the temporomandibular joints. The sella turcica appears normal.

"Mastoid Areas: Examination in the Law and the Stenver projection shows a moderate and symmetric cellular development. The cells are pneumatic on

both sides and no fracture or bone destruction is demonstrable. The petrous ridges appear intact."

On December 5 an operation was done which consisted of exploration of the facial nerve and internal and external neurolysis of the facial nerve.

The usual postauricular incision was made, the mastoid periosteum elevated and the cortex of the mastoid exposed. The tip of the mastoid was located and the incision extended anteriorly in order to expose the parotid gland. In the first stage of the operation the facial nerve was exposed in the mastoid portion of the temporal bone after the technic described by Tickle and was found to be uninjured in this region. The nerve was then followed anteriorly into the substance of the parotid gland. Here the nerve was embedded in the dense scar tissue. There was no actual loss of continuity except for the lower branch of the facial nerve (mandibular), which had been torn. The nerve was dissected free of scar tissue, and an internal and external neurolysis was done. The wound was closed, and the patient was returned to the ward in good condition.

Fig. 6 (case 9).—The small wound of entrance in the left cheek (indicated by arrow) and the complete peripheral paralysis of the right facial nerve.

The postoperative course was uneventful, and the wounds healed satisfactorily. Physical therapeutic treatment was instituted, and the patient was fitted with a support for the corner of the mouth. He was evacuated to the zone of the interior for further treatment and observation.

CASE 10.—A private, aged 24, was wounded in action on March 10, 1945, on Luzon, Philippine Islands. A large metallic foreign body struck the patient just posterior to the tip of the mastoid portion of the temporal bone, then passed under the periosteum and made its exit through the left cheek, just anterior to the ramus of the left mandible. The initial examination revealed that the patient had a complete left peripheral facial palsy and a fracture of the ramus of the left mandible. Roentgen studies made on March 20 by Major W. A. Evans revealed the following:

"Facial Bones: Examination in the nose-forehead, the extended chin and the left oblique projection shows comminution of the mandible involving the left ascending ramus, with a fracture through the base of the coronoid process and with extensive comminution at the base of the condyloid process. Fixation wires

are seen about the teeth. An incomplete linear fracture is noted in the left malar eminence, but there is no deformity in this area, and the paranasal sinuses are pneumatic. No foreign body is seen. The mastoids show no evidence of foreign body or infection."

The right and the left membrana tympani were normal in appearance. There was no evidence of perforation or infection. Galvanic and faradic studies of the left (facial) nerve revealed a good response to both currents.

In view of the absence of infection, as observed both clinically and roentgenologically, and the fact that the nerve responded well to the electrical tests, it was decided that the nerve itself had not been injured. It was felt that the facial paralysis was secondary to tissue edema and that the prognosis for recovery of function was good. The patient was given physical therapy to the left side of the face. The fractured jaw was adequately treated, and the patient was evacuated to the zone of the interior.

CASE 11.—A sergeant, aged 24, was wounded in action on Feb. 8, 1945. He received multiple shell fragment injuries of the right side of the face and neck, as the result of which he sustained a compound comminuted fracture of the right ascending ramus of the mandible and an injury of the right parotid gland. There was complete peripheral paralysis of the right facial nerve as the result of this injury. The patient was treated in the forward area and was evacuated to this hospital, arriving March 2.

At the initial examination the patient's condition was good. The external wounds were well healed. The fractured mandible had been reduced, and the teeth were in good occlusion. Roentgen studies made on March 5 by Major W. A. Evans revealed the following:

"Facial Bones: Examination in the nose-forehead, the extended chin, the right lateral and the right oblique projection shows a fracture involving the right ascending ramus of the mandible at the base, below the condyloid process. There is no appreciable displacement or disalinement of the fragments, and normal relations are maintained at the temporomandibular joints. The other facial bones appear intact, and the paranasal sinuses are pneumatic, except for some clouding of the ethmoid cells on the left. Numerous metallic fragments are seen in the soft tissues about the right ascending ramus of the mandible.

"Mastoid Areas: Examination in the Law and the Stenver projection shows a moderate and symmetric development. All the cells appear pneumatic with the trabeculae intact, and no fracture or inflammatory involvement is demonstrable. The petrous ridges appear intact, and normal relations are maintained at the temporomandibular joints."

Galvanic and faradic tests made for function of the facial nerve indicated a complete loss of response to the faradic current. In view of the extensive injury to the facial nerve in the substance of the parotid gland it was reasoned that there would be no benefit from exploration or attempted anastomosis. Some type of facial plastic operation would be necessary, and the patient was evacuated to the zone of the interior. In this patient there was no evidence of injury to the mastoid.

CASE 12.—A private first class, aged 23, was wounded in action on Feb. 8, 1945. A shell fragment approximately 1 by 0.7 cm. made its entrance at the angle of the right jaw and then lodged at a point close to the tip of the right transverse process of the second cervical vertebra. The patient was admitted to this hospital on March 10 after having received preliminary treatment in the forward area.

On his admission to the hospital the preliminary examination revealed that the patient's general condition was good. Roentgen examination made on March 10 by Major W. A. Evans revealed the following:

"Mastoid Areas: Examination in the Law and the Stenver projection shows a moderate and symmetric cellular development. All the sinuses appear pneumatic with the trabeculae intact, and no fracture or inflammatory involvement is demonstrable. There is, however, a metallic fragment 1 by 0.7 cm. lying in the soft tissues medial to the tip of the right mastoid process, with a minute metallic fragment nearby, posterior and medial to the condyloid process of the mandible. The fragment lies close to the tip of the right transverse process of the atlas, and there may be a limited injury to its right transverse process.

"Facial Bones: Examination in the nose-forehead, the extended chin and the right oblique projection shows no fracture of the mandible or other facial bones, and normal relations are maintained at the temporomandibular joints."

The external wounds were well healed. The patient had complete peripheral paralysis of the right facial nerve. Galvanic and faradic tests of the function of the facial nerve gave good response to both currents and indicated that recovery of nerve function was occurring. The problem then was one of proper physical therapeutic management and of awaiting the restoration of function. The patient was evacuated to the zone of the interior for further treatment.

COMMENT AND SUMMARY

The number of casualties that sustain injury to the mastoid area and facial nerve are few. There were 12 patients who arrived at this hospital with this type of injury, of a total of 4,400 battle casualties requiring surgical treatment. This is a small number from which to draw conclusions and to attempt to lay down rules of therapy. However, it is felt that many valuable lessons have been learned.

Within the temporal bone are present many vital structures in a small area, so that the entrance of a foreign body may produce much damage. Often there are fractures of the dural plate, the lateral sinus plate, and/or injuries to the dura or lateral sinus. In battle injuries infection occurs rapidly. The presence of blood, devitalized tissue, damaged bone and foreign bodies forms ideal conditions for the growth of bacteria. The combination of infection plus injury to important anatomic structures could easily lead to dangerous intracranial or systemic infection. That this did not occur attests to the efficiency of the sulfonamide drugs and to the administration of penicillin in the preoperative period.

Clinically few symptoms may be present that would indicate the extent of damage. There was purulent drainage from the middle ear and the mastoid area in all patients. The real extent of injury is only learned at the operating table.

The roentgen rays are invaluable in the study and analysis of this type of injury, and I believe that the importance of the roentgenograms has been ably summarized by Major W. A. Evans whom I quote:

The X-Ray examination of the patients who have sustained injury to the mastoid area requires detailed exposures of the area in at least two projections. An adequate examination may be expected to reveal accurately the presence and location of foreign bodies, evidence of fractures, and, in most cases, the presence of infection. Fractures tend to be more extensive and inflammatory changes more advanced than is apparent in the roentgen study. Occasionally inflammatory changes are present in the mastoid cells when there is no evidence of infiltration in the X-Ray films.

These patients had received excellent care from the moment of injury, and were rapidly evacuated to areas in which skilled surgical treatment was available. However, my colleagues and I have noted that too much reliance is placed on the chemotherapeutic agents. Chemotherapy cannot take the place of surgical intervention nor will it substitute for incomplete surgical treatment. The term "débridement" or "partial débridement" is often an excuse for incomplete surgical treatment.

Our cases illustrate that the extent of injury was always greater than we had anticipated. Hence it is believed safe to postulate that when surgical treatment is begun the surgeon should be ready to do a complete mastoid operation, to follow the injury wherever it may lead.

When the injury is localized to the mastoid a complete mastoidectomy should be done. If the middle ear is involved as well, then a tympanomastoidectomy is indicated.

When a foreign body is present in the mastoid area simple removal of the foreign body is not adequate therapy. At the time of the removal a mastoidectomy should be done which will allow ascertainment of the extent of injury and removal of the foreign body and injured bone structures. This will result in rapid recovery. Otherwise a long period of disability may be anticipated, with continued suppuration and the ever present threat of a dangerous complication.

Injuries of the facial nerves pose a problem of diagnosis and treatment. The electrical tests of nerve function give valuable information as to the prognosis. When patients give good response to the faradic current recovery may be expected. The technics of Ballance and Duel and Tickle were utilized. A graft of the facial nerve was done in the presence of infection in 1 case, and recovery ensued. The postoperative course while awaiting recovery is a difficult period for both physician and patient. Physical therapy is important in order to prevent atrophy of the facial muscles.

Chemotherapy and partial surgical treatment are not adequate therapy for the types of wounds indicated in this paper. Chemotherapy and complete surgical treatment will result in rapid recovery.

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ABNORMAL COURSE OF THE FACIAL NERVE IN THE FALLOPIAN CANAL

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IN THE anatomic and otologic textbooks the course of the descendant part of the facial nerve is stated to be constant, and during an operation it may be localized simply by running a straight line from the most overhanging point of the lateral semicircular canal to the stylomastoid foramen. Bridgett [1] proceeds in the following manner: A probe inserted into the mastoid notch will point directly toward the stylomastoid foramen; after the exenteration of the cells in the tip of the mastoid process, however, this notch can be seen as a salient ridge, the crista digastrica at the floor of the cavity. If this ridge is followed anteriorly as far as the posterior wall of the acoustic meatus it will be seen to end at the stylomastoid foramen, from which a thin, bony lamina passes upward, toward the lateral semicircular canal, indicating the course of the nerve. The intersecting point of these two lines is called the junctio inframastoidea. Batson,[2] looking up the stylomastoid foramen along the saggital plane in just the same manner, pointed out that in the frontal plane the foramen and the vertical part of the facial canal are placed perpendicularly medial to the suture between the tympanic and mastoid parts of the temporal bone—the tympanomastoid suture. As a rule this suture is visible, but at any rate it can always be felt by means of a probe. "The facial nerve is not found posterior to this suture. It may be slightly anterior."

If this rule were without any exception, it would have been a rule of great practical importance. Hawley,[3] however has in three hundred dissections four times found the nerve displaced 3 or 4 mm. posterior to this plane, and in 1 case the nerve, terminating in a blind pouch, sent only a slender string in the normal direction.

On the basis of one hundred and twenty-five operations on the facial nerve performed in the manner of Ballance and Duel, I am able to confirm the statements made by Hawley, as in no less than 7 cases more or

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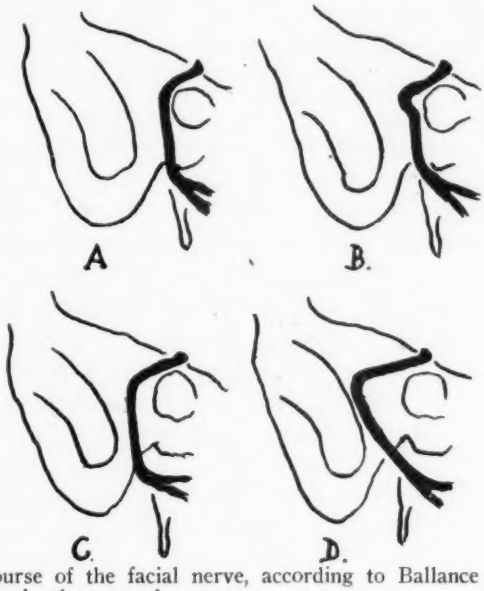
1. Bridgett, F. A.: Monatschr. f. Ohrenh. **57**:187, 1923.

2. Batson, O. V.: Surgical Anatomy of the Temporal Bone, in Nelson's Loose-Leaf Surgery of the Ear, New York, Thos. Nelson & Sons, 1938.

3. Hawley, C. W.: Illinois M. J. **41**:116, 1922; Zentralbl. f. Hals-, Nasen- u. Ohrenh. **1**:156, 1922.

less considerable deviations from the normal course could be demonstrated. These variations manifested themselves in three different ways:

1. Between the stylomastoid foramen and the origin of the chorda tympani the nerve pursued a normal course, but just proximal to the chorda it formed a posteriorly convex curve before resuming its usual course forward and medially to cross the promontory (*B* in figure). An arrangement like this I have met with three times, a fact which should be borne in mind, because the most prominent point of this



A, normal course of the facial nerve, according to Ballance and Duel. *B*, *C* and *D*, variations in the normal course.

posteriorly convex curve is directly distal to the most overhanging part of the lateral semicircular canal, a reason why this portion of the nerve is especially exposed to the danger of being injured during a simple mastoidectomy.

2. In 3 cases the nerve was displaced 2 to 4 mm. dorsally all along its vertical course; it did not pass along the posterior wall of the acoustic meatus, but deep in (medial to) the threshold cells (between the posterior wall of the osseous acoustic meatus and the sinus sigmoideus).

This variation too is important, as during the process of exenterating the threshold cells the nerve may become injured. A danger signal is a suddenly occurring arterial hemorrhage during the excochleation, coming from the stylomastoid artery; should there be such an occurrence, a careful investigation should immediately be instituted.

3. The anomalies of the course pursued by the nerve, described under 1 and 2, may be combined, and an extreme instance of this anomaly was seen in the following case:

In a woman of 29 suffering from partial Bell's palsy, decompression of the facial nerve had been performed elsewhere, and the description of the operation reads as follows: "At a short distance below the level of the floor of the external acoustic meatus the nerve, having in its previous course been of normal size and development, suddenly becomes a mere thread, while at the same time the lumen of the canal is considerably diminished. At last both nerve and canal apparently disappear altogether, making it extremely difficult to orientate oneself. In the meantime most of the facial ridge has been chiseled away. The upper portion of the threadlike nerve proves to have been torn in two, and the canal at the site of injury is so narrow that a Zaufall hook cannot be introduced into it but only the tip of a labyrinth probe. The bone of this part is completely firm and hard, and apart from the atrophy the nerve and the nerve sheath have shown nothing abnormal. The operation is postponed." Complete facial palsy developed in direct conjunction with the operation.

I was consulted and discovered that the operator, when performing the decompression, had followed the chorda tympani and resected the descendant part of the facial nerve. The proximal end of the facial nerve was found as far away as at the anterior margin of the sinus capsule (*D* in figure), the intervening piece of the nerve between this place and the origin of the chorda tympani having been removed during the exenteration of the threshold cells. A nerve graft was inserted in the manner of Ballance and Duel, and the patient recovered her mobility.

These discoveries made at operations fall into line with the results of the investigations made by Hawley and prove the untenability of the allegation expressed in the anatomic and otologic textbooks to the effect that the facial nerve is never found dorsal to a frontal plane through the tympanomastoid suture. These anomalies of the course pursued by the facial nerve should be borne in mind whenever an operation on the mastoid process is performed, particularly in cases of radical exochleation of the threshold cells, and the surgeons who chiefly perform operations on the facial nerve by the method of Ballance and Duel should, when confronted with the problem of looking up the stylomastoid, adapt the technic accordingly. Once the nerve has been found, spectacles with magnifying glasses should be used during the decompression of the nerve, which must be followed upward step for step and denuded to its full breadth. If this precaution is omitted and the nerve is promptly looked for at its "official site," sooner or later it will inevitably become injured.

SUMMARY

In one hundred and twenty-five operations on the facial nerve by the method of Ballance and Duel, the nerve was in 7 cases found to be completely or partly displaced, so that it was dorsal to a frontal plane through the tympanomastoid suture, the latter indicating the normal course of the nerve. The surgical significance of this discovery, which confirms earlier investigations made by Hawley, is mentioned.

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PENICILLIN IN THE ORAL THERAPY OF VINCENT'S ANGINA AND ACUTE FOLLICULAR TONSILLITIS

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THIS preliminary report is not intended as a completely scientific study of a problem about which much is still being written but rather as an attempt from an overseas theater to add some material for the evaluation of penicillin as used in the oral therapy of tonsillar infections. Only two conditions will be dealt with in this report: Vincent's angina and acute follicular tonsillitis.

There is still much discussion as to the etiologic nature of Vincent's angina, and poor oral hygiene, local irritation, debilitating disease and other conditions have been called predisposing factors. The question has been raised as to whether the spirochetes and the fusiform bacilli of Vincent may not be pleomorphic forms of the same organism. However that may be, the manner in which lesions showing the presence of these organisms respond to treatment with penicillin can leave little doubt that Vincent's organisms are certainly associated with the disease if they do not directly cause it.

Of interest in the military field is the increase in the rate of Vincent's infection per thousand men per month from 2.23 in 1940 to 3.33 in 1944, almost 50 per cent. Overseas the rate has gone from 1.03 in 1942 to 2.60 in 1944. Owing to the necessity of hospitalization for isolation in the Army, this has produced a tremendous loss of man days per month under older methods of treatment. Under treatment with some one or combination of the following methods the hospital stay has averaged two weeks: irrigation with sodium perborate or potassium permanganate; topical applications of silver nitrate, 5 or 10 per cent chromic acid, trichloroacetic acid, copper sulfate, 10 per cent neoarsphenamine or arsphenamine, or bismuth subnitrate.

In June 1944, at the suggestion of Dr. Gordon Hoople, my associates and I adopted the following procedure for the treatment of Vincent's angina. A smear was taken prior to the initiation of treatment to confirm the clinical diagnosis. The ulcer was sucked to the raw, bleeding surface; a 3 per cent solution of hydrogen peroxide was sprayed into the ulcer cavity, followed by insufflation of sodium perborate powder. This was done twice daily. The results in 46 cases are summarized in the upper half of the accompanying table. Just over three days' treatment was required for a smear to be obtained which

would show no spirillas or fusiform bacilli. The total hospitalization averaged just under seven days.

In November 1944 MacGregor and Long [1] published a preliminary report on the results obtained with penicillin pastilles in oral infections. Their pastilles were composed of calcium penicillin in gelatin with 0.1 per cent Nipagin added as a preservative.[2] They used 250 unit pastilles in Vincent's and 500 units in other tonsillar infections. They felt that pastilles containing 1,000 or more units might prove even more effective but were prevented by limitations of supply from carrying out studies with these higher doses. The method used required the patient to dissolve one pastille after another in his mouth throughout the day. In 25 cases of Vincent's angina described in their paper the clinical and bacteriologic responses were as follows: All patients were asymptomatic in twenty-four hours; ragged sloughs were gone in forty-eight hours and treatment was stopped in five days. In twenty-four hours spirochetes were absent and the number of fusiform bacilli was reduced; in forty-eight hours the number of fusiform bacilli was reduced, or these forms were absent; in seventy-two hours both spirochetes and fusiform bacilli were absent.

In setting up our method of treatment there were several problems: One was that the pastilles and calcium penicillin were unavailable, another, that the 100,000 unit ampule of sodium penicillin used was of such small volume that 500 or 1,000 unit doses were almost microscopic and difficult to handle with the facilities available. It was decided to divide the 100,000 units into ten doses of 10,000 units each and place these amounts in gelatin capsules. During part of the study the penicillin salt was combined with approximately 0.13 Gm. of microcrystalline sulfathiazole powder for use in some of the cases of tonsillitis, about which I shall say more later. The work of Bigger [3] and Ungar [4] seems to indicate that there is a synergistic effect between penicillin and the sulfonamide compounds.

The patients were instructed to permit the penicillin capsule to dissolve in the buccal sulcus and were given 1 capsule every two hours for ten doses. In the 25 cases studied the clinical and bacteriologic responses were as follows: All patients were asymptomatic in twenty-four hours, ragged necrotic ulcers were replaced by smooth, shiny membrane in twenty-four hours and complete healing, with no recur-

1. MacGregor, A. B., and Long, D. A.: The Use of Penicillin Pastilles in Oral Infections, Brit. M. J. **2**:686 (Nov. 25) 1944.

2. Nipagin is produced by the Goldschmidt Chemical Corporation, New York.

3. Bigger, J. W.: Synergic Action of Penicillin and Sulfonamides, Lancet **2**:142 (July 29) 1944.

4. Ungar, J.: Synergistic Effect of Paraminobenzoic Acid and Sulfapyridine on Penicillin, Nature, London **152**:245 (Aug. 28) 1943.

rences, took place in five to seven days. All smears were free from both spirillas and fusiform bacilli at the end of the tenth dose, or twenty-four hours after the beginning of the treatment. In 2 cases smears showed absence of spirillas and fusiform bacilli at the end of the sixth dose; in three, after the eighth dose, and in 1, after the ninth. The lower half of the table shows the results of the penicillin method of treatment: one day's treatment and five days' total hospitalization and that despite the fact that in the earlier cases the patients were retained in the hospital longer than was actually necessary in order to study the possibility of recurrences. When it was found that all ulcers healed completely in five to seven days, the patients were discharged on the completion of one day's treatment when the smear was reported negative. It is my opinion that military patients with Vincent's infection no longer require hospitalization at all, since most of them have

Comparison of Methods of Oral Treatment for Vincent's Angina

Treatments	Days	Total Days of Hospitalization
46 Cases in Which the Suction, Hydrogen Peroxide, Sodium Perborate Treatment Was Given Twice Daily		
6+	3+	7—
25 Cases in Which Capsules Containing Penicillin Sodium Were Given, One Every Two Hours		
10	1	5

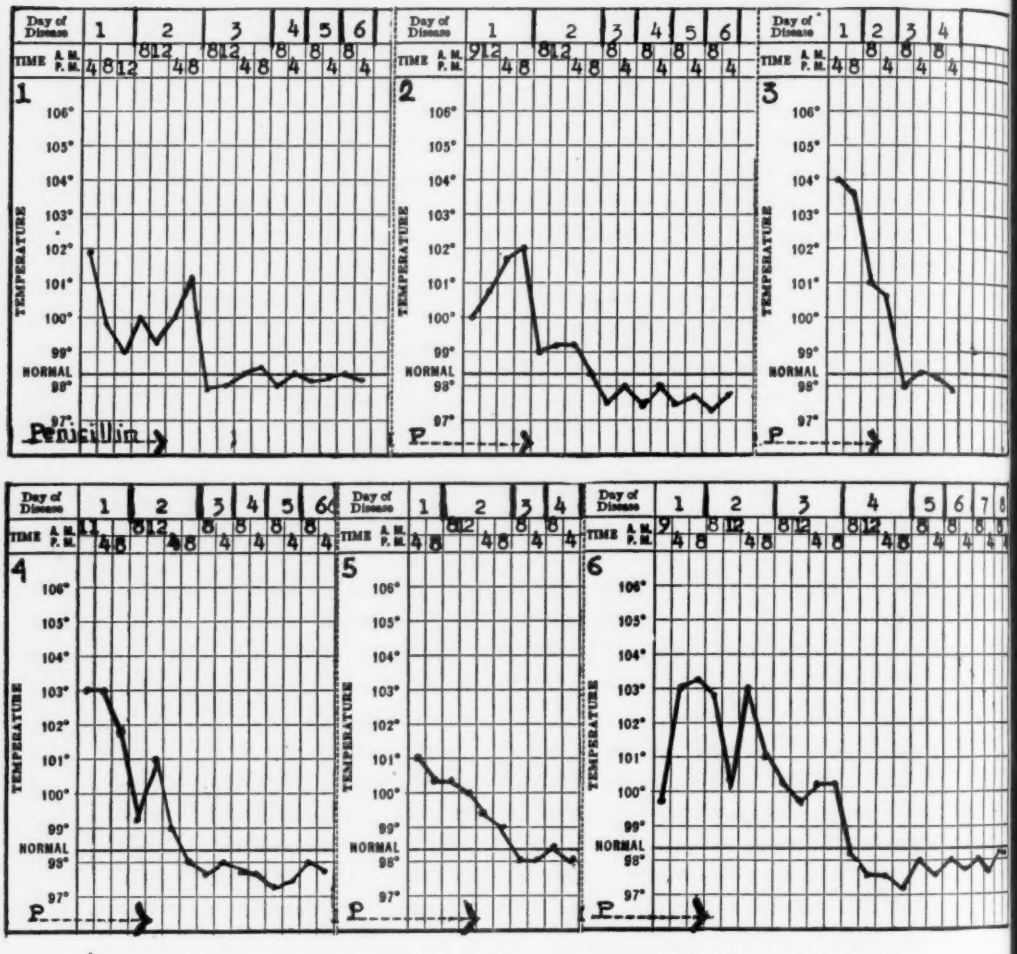
had the condition for three to seven days prior to admission to the hospital and the additional twenty-four hours necessary for cure would not greatly increase the hazards of transmission.

One case may be of interest in that both old and newer methods of treatment were used for different episodes: On Nov. 7, 1944 the patient had Vincent's angina and received the suction, hydrogen peroxide, sodium perborate treatment twice daily. Three days were required to obtain a smear that showed absence of spirillas and fusiform bacilli. On Feb. 12, 1945 the patient had another episode of Vincent's infection, and eight doses or sixteen hours' treatment with penicillin were required to obtain a smear free from spirillas and fusiform bacilli.

In addition to the increased rapidity of cure, the great decrease in the patient's discomfort obtained by substituting a simple capsule, to be dissolved in the mouth, for a gagging, occasionally painful suction and multiple spray should not be overlooked.

Thirty-seven patients with severe acute follicular tonsillitis were treated; patients with mild involvement were not referred to the clinic for diseases of ear, nose and throat. Typical temperature graphs as recorded in 5 unselected cases are shown in the chart (graphs 1 to 5).

Penicillin capsules were given as in the cases of Vincent's infection, one every two hours. Clinically, every patient felt considerable relief from soreness of the throat in twenty-four hours or less, and the temperature fell to normal within twenty-four hours after the completion of the penicillin course. This was so consistent that when the fall of



Typical temperature graphs in 6 cases of acute follicular tonsillitis. Arrow heads indicate the time at which penicillin therapy was completed.

temperature did not occur, a cause for the elevation other than the tonsillitis was suspected. One case serves to illustrate:

There was an onset of sore throat two days prior to admission to the hospital, with the body temperature ranging to 103.2 F. on the first hospital day (graph 6 in chart). Culture of material obtained from the throat showed Diplococcus pneumoniae, hemolytic Staphylococcus aureus and nonhemolytic Staph. aureus.

The patient was given ten capsules containing penicillin sodium, 10,000 units each. Symptomatically and clinically the throat was improved at the end of twenty-four hours, and the culture then revealed only hemolytic Staph. aureus. After forty-eight hours the patient's temperature continued elevated, and he complained of cough and thoracic pain. Clinical examination and roentgenograms revealed an early stage of pneumonia (roentgenologist's report: "There are linear infiltrative densities extending from the left hilar region into the base of the left lung, which represent an early or minimal inflammatory process"). This cleared rapidly with appropriate treatment, the throat remaining normal throughout.

Five patients were given two courses of treatment with the capsules. This procedure was followed especially in the early days of treatment when the response was not yet clear. There were 2 cases in which *early* peritonsillar abscess, manifested by dysphagia, unilateral peritonsillar swelling and a slight degree of trismus, subsided with no other treatment. There were 2 cases in which peritonsillar abscess necessitated incision and drainage after the course of treatment with penicillin. It is felt that beyond a very early stage the oral administration of penicillin will not affect the peritonsillar abscess.

Ten of the 37 patients received the penicillin-sulfathiazole combination already mentioned. This therapy was discontinued because traces of sulfathiazole were found in the blood after the tenth dose and because the clinical response did not appear improved by the addition of the sulfathiazole.

In the cases of acute tonsillitis hospitalization lasted approximately two weeks without recurrence being observed, which should have provided a sufficient interval for the current episode to be considered cured.

SUMMARY

Twenty-five patients were treated for Vinicent's angina with penicillin sodium in gelatin capsules, 10,000 units each, one being given every two hours for ten doses. Cure was obtained in twenty-four hours.

Thirty-seven patients were treated similarly for acute follicular tonsillitis, with the body temperature falling to normal in twenty-four hours and marked symptomatic relief; cure was obtained in three to five days.

Further study is required to determine (*a*) the least number of doses of penicillin necessary for cure and (*b*) the smallest dose that will yield results as good as those obtained with the doses mentioned.

Charles K. Brauer, M.D., Jersey City, N. J., assisted in the clinical studies.

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RHINITIS AND EUSTACHIAN SALPINGITIS

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THE TERM "sinusitis" has a definite meaning. There is a tendency, however, to use it as a "grab bag," into which all problems referable to the nose are thrown. It is just as dangerous to put the word into the vocabulary of the enlisted man as is the term psychoneurosis. Preferably, it should be used with care and only after definite evaluation. Persistent purulent discharge from the upper part of the nose following the usual period of a "cold in the head" is the deciding point, a condition which occurs in only a relatively small percentage of personnel at the Orlando Army Air Base. The symptom complex of headache, nasal obstruction and anterior or posterior discharge of nasal mucus is usually indicative of acute catarrhal rhinitis and not of sinusitis.

Cultures of discharges of nasal mucus from normal-appearing nasal passages were taken at the Orlando Army Air Base during a study of acute and convalescent rheumatic fever as well as various types of arthritis. One important finding in these cultures was the presence of hemolytic or nonhemolytic Staphylococcus aureus or Staphylococcus albus in about 50 per cent of the cases. Hemolytic or nonhemolytic streptococci were found in the other 50 per cent.

Another important factor is found in the complex group of extrinsic (air borne) or intrinsic (blood borne) organic or inorganic allergens which irritate the sensitized mucosa, setting up a visible or an invisible allergic activity.

A few words may be said on the meaning of the terms rhinitis, aero-sinusitis and aero-otitis. The definition of inflammation is "a morbid condition characterized by redness, pain, heat and swelling." Rhinitis is defined as inflammation of the nasal mucous membrane. The behavior of the important tissues of the nasal passages will be explained in a later paragraph. The manner in which the pseudo signs and symptoms of inflammation are presented to the rhinologist will be outlined. The pathologic and clinical concepts of inflammation are not comparable to the action of the nerves, glands and vessels of the nasal mucosa under the stress of extrinsic and intrinsic irritants. A disturbance in the fac-

From the Eye, Ear, Nose and Throat Department, Army Air Force Regional Station Hospital, Orlando Army Air Base, Orlando, Fla.

tors of the nasal mucous membrane takes place on the ground, before the flying personnel take to the air to become subject to the effects of altitude and cold. Aero- is defined as air, aerial, gas or gases. Air or gas has as much to do with aerosinusitis or aero-otitis contracted in flight as it has to do with riding in a hilly country or up a mountain road. The standard terms of rhinitis, salpingitis and otitis media are sufficient to cover the problem and conform to military medical terminology.

In the recent papers relating to diseases of the nose and ears,[1] attention has been called to many of the present aids in the hospital analysis

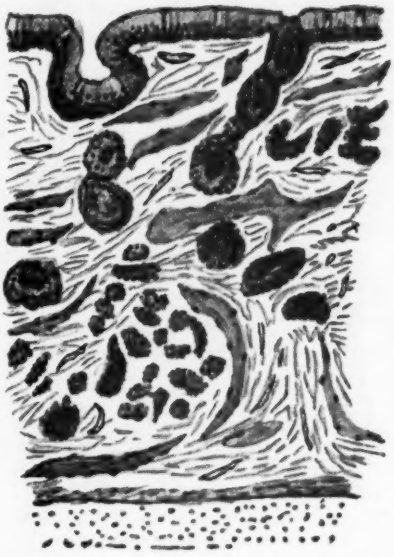

Fig. 1.—Elements of the mucous membrane common to nose and eustachian tubes: nerves, musculovascular sinus beds and mucous glands and lymphatics.

of these conditions. However, a flight surgeon normally is not equipped in the theater to engage in many of the finer approaches.

The method of treatment to be considered in this paper has its basis in the clinical behavior of the microscopic anatomic structures, which are the most important elements in the lining of the bony cartilaginous passages of the nose and eustachian tubes. Therapy need not be based on nasal packs, sinus punctures, irrigations, ionization, cauterizations or displacement methods. In the treatment of the nasopharynx and eustachian tube, excellent results can be attained without the use of

1. Wright, R. W.: Aero-Otitis, Air Surgeon's Bull. (no. 6) **1**:8 (June) 1944. Spiegel, F. S.: Aero-Otitis, ibid. **1**:9 (June) 1944. Bell, J. J.: Aero-Otitis. ibid. **1**:10 (June) 1944.

inflations by the Politzer bag, catheter, Valsalva and pressure methods or dilations by finger or bougie.

ANATOMIC CONSIDERATIONS

The elements of the mucous membrane (fig. 1) common to nose and eustachian tubes are (1) nerves, (2) fine superficial and deep cavernous musculovascular sinus beds and (3) mucous glands and the lymphatic vessels. The thin layer of mucus which covers them is slowly directed by the moving cilia and deposited into the nasopharyngeal space. This mucus is constantly being formed by the mucous glands in the normal state and becomes greatly increased in allergic activity.

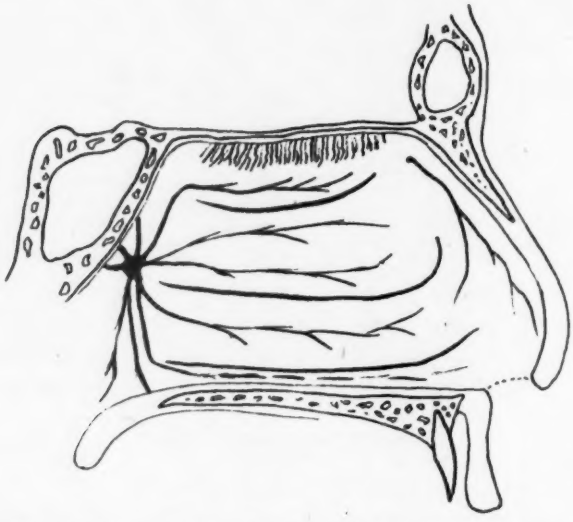

Fig. 2.—Nerve supply of the nasal mucous membrane.

The nerve tissue plays a dominant role (fig. 2). All nerve tissue has an upper, or central, neuron and a lower, or peripheral, neuron (fig. 3). The lower sensory and motor neurons send antennae to the epithelium of the mucous membrane, which contact the chemical and bacterial irritants of the environment, as well as communicating nerves to the mucous glands and musculovascular sinuses. The upper sensory and motor neurons of the basal nuclei and cerebrum influence or can be influenced by the behavior of the lower ones. These tissues are all subject to the toxic and hormonal content of the blood and hence are affected by disease and normal body processes as well as by the independent inherent behavior of the tissue cells and fluids.

A number of situations and variations (fig. 4) may affect the three functional tissues. The nerve tissue, mucous glands and vascular sinuses are represented. Activity is indicated by the darkness of the color.

1. Normal-acting elements are indicated in figure 4 *A*.

2. The nerve bed may be irritable in a localized area while the blood bed and mucous glands are quiet (fig. 4 *B*).

3. A sensitive nerve bed may be present while the mucous glands are stirred into activity in a localized area, with the vascular bed remaining quiet (fig. 4 *C*).

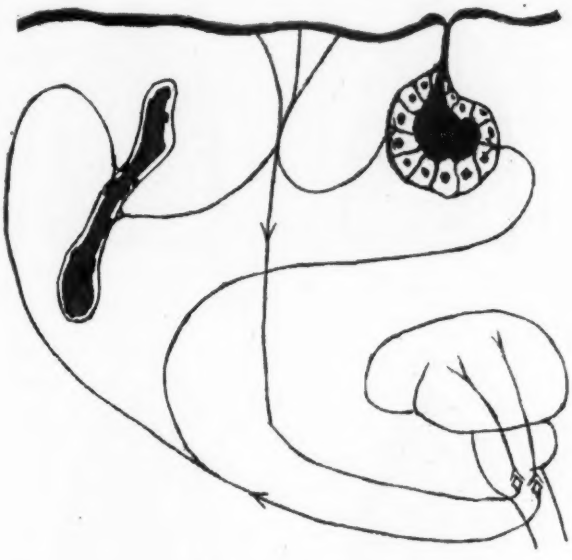

Fig. 3.—Lower and upper neurons.

4. All three tissues may be hyperactive (fig. 4 *D*).

5. A sensitive nerve bed may be present with an irritable blood sinus bed and a quiet mucous gland bed, with obliteration of the airway (fig. 4 *E*).

6. The nerve tissue may be quiet and the mucous glands and musculovascular bed active (fig. 4 *F*).

7. The mucous glands may be actively secreting while the nerve and blood beds are quiet (fig. 4 *G*).

8. The vascular beds may be engorged, obliterating the airway, while the nerve and mucous gland tissues are quiet (fig. 4 *H*).

The mass of tiny, compact, nerve antennae which form the nerve bed of the nasal and eustachian mucosa seems to play the dominant role in the behavior of the membrane, particularly in relation to the patient's complaints, especially of headache and earache. Lymphatic channels, burdened with intercellular fluid, increased the depth and firmness of the mucous membrane, decreasing the patency of the airway. This intercellular fluid persists in proportion to the degree and time of vascular-sinus bed engorgement. When one places such a tissue in a variety of cartilaginous and bony nasal passages as is found in the fusion and

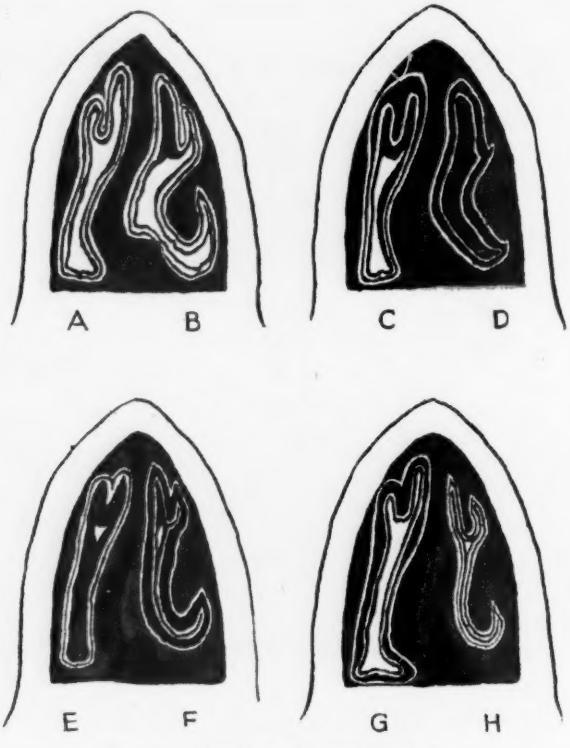

Fig. 4.—Situations in nasal mucosa.

growth of bone and cartilage in prenatal and postnatal life, one can more readily comprehend the varieties of physiologic and clinically normal noses.

A deviation of the nasal septum only distorts the nasal airway. An obstruction caused by congestion in a narrow rigid passage permits pronounced pressure from thickening of the mucous membrane, with squeezing and irritation of the nerve endings in that area. This may give rise to tender, or "trigger," areas, which initiate a general cephalalgia or a pain localized in any area in the face or head, depending on the patient's

threshold for pain. A submucous resection will allow only bony clearance for ventilation or for future engorgement of these painful areas, especially if they are near the ostiums of the various sinuses or between the septum and the middle or superior turbinate bones. These sensitive areas are also found in adequately ventilated passages free from bony or vascular obstruction and vary with fatigue. Such irritability may be due to a variety of factors such as monotony, long working hours, unhappy surroundings, improper food and drink, unsuitable working conditions, lack of hobbies and many other psychosomatic factors.

SIGNS AND SYMPTOMS

The procession of signs and symptoms are usually not orderly. They vary in their onset, severity, timing and complications. The usual order of situations presented are the same with rhinitis (head cold) and allergic conditions of the nose.

I. Acute catarrhal rhinitis

A. Situation (fig. 4 *B*)

1. Sensitiveness to the flow of air through the nose
2. Sneezing
3. Headache

B. Situation (fig. 4 *C*)

4. Anterior or posterior discharge of mucus which further irritates the nerve bed
5. Purulent discharge may or may not develop; pus is usual in a head cold and less frequent with an allergy

C. Situation (fig. 4 *D*)

6. Stuffiness and obstruction to breathing
7. Pressure, which in turn increases irritation and formation of mucus

Thus a vicious circle is established and will continue until the link of nerve irritation is broken in the chain of situations of signs and symptoms.

II. Acute catarrhal eustachian salpingitis

A. Situation (fig. 4 *B*)

1. Itchiness in the ear
2. Intermittent or steady ache or pain deep in, below or in front of the external auditory canal

B. Situation (fig. 4 *C*)

3. Stuffiness in the ear, caused by increased mucus
4. Loss of hearing in certain higher frequencies, above 2,048

C. Situation (fig. 4 *D*)

5. An increase in the "plugged ear" feeling, from congestion or congestion and lymphatic stasis
6. An increase in pain or ache
7. Loss of hearing in the higher frequencies, above 2,048, and often a loss in the conversational or lower frequencies, below 2,048
8. Tympanic membrane signs, in order of severity
   (*a*) No signs
   (*b*) Dilatation of a few blood vessels on the flaccida and malleolus
   (*c*) Fluid and air bubbles in the middle ear
   (*d*) Ecchymoses in the membrane
   (*e*) A dull red thickening of the flaccida, which extends down the malleolus and around the annulus as congestion increases
   (*f*) A general obliteration of all landmarks by a dull red-gray thickened membrane

TREATMENT

The object of the treatment is to return the three elements to a normal state. Emphasis is placed on the control of the nerve tissues at the earliest opportunity.

I. Medicinals

a. One capsule containing codeine sulfate, ½ grain (0.03 Gm.), atropine sulfate 1/400 grain (0.00015 Gm.), and acetylsalicylic acid, 10 grains (0.65 Gm.), after breakfast, after supper and at bedtime, for a few days (codeine and acetylsalicylic acid depress the upper and lower neurons; atropine diminishes the activity of the mucous gland and relaxes involuntary muscle that is responsible for vascular congestion and escape of serum and cells into the tunica propria of the nasal and eustachian mucosa).

b. One capsule contain[illegible]lfadiazine, 5 grains (0.32 Gm.), and sodium bicarbonate, 10 grain[illegible] (0.65 Gm.), taken after each meal and and 2 at bedtime (tolerance of sulfonamide drugs by the patient should guide the dosage; this will inhibit the sulfonamide-susceptible bacteria in the noseways and eustachian tubes and lessens possibility of such complications as suppurative sinusitis, nasopharyngitis, otitis media, laryngitis, meningitis and tracheobronchitis).

This form of medication (*a* and *b*) has been used since the days of Prontosil (azosulfamide). It has been successful in the treatment of

patients in the eye, ear, nose and throat outpatient department during the past twenty-two months. Recent articles [2] deal with similar problems.

c. Intramuscular doses of 5 mg. of thiamine hydrochloride are given daily for tardy congestion of the eustachian tube.

II. Local therapy.

a. Gentle application to the sensitive areas in the nasal mucosa of 0.5 per cent cocaine hydrochloride solution, followed by 0.5 per cent atropine sulfate solution (this requires a small cotton-tipped steel applicator, a nasal speculum, a head mirror and a lamp).

b. Gentle treatments of the pharyngeal eustachian tube with application of solutions in paragraph *a,* followed by applications of 1 per cent ephedrine sulfate, 5 per cent aqueous solution of merbromin or 1 per cent silver nitrate. A 70 per cent solution of alcohol carefully applied produces a warm, stimulating sensation that radiates from the nasopharynx to the external auditory canal.

III. General treatment

A. One glass of water hourly while awake; drink water only for liquids and beverage

B. Eat heartily of substantial foods, without desserts

C. Avoid blowing the nose; rather inspirate and swallow

D. Avoid theaters and crowds

E. Go to bed early for rest or sleep

F. Work as usual

IV. Allergic therapeutic considerations

Allergic conditions in the nose and eustachian tubes present further study.

A. The most frequent blood-borne allergens are derived from:

1. Diseases of the gastrointestinal tract, the cardiovascular system and the renal organs, anemia and other fatiguing diseases
2. Liquid intake such as fruity juices, synthetic drinks, tea, coffee, milk, cocoa, malt, chocolate and alcoholic beverages
3. Food such as eggs, juicy fruits, pie, cake, pastry, ice cream, potatoes and bread

B. The most frequent air-borne allergens are dust, bacteria (staphylococci, streptococci and fungus); pollen protein from trees,

2. Whalen, E. J.: Sulfonamide Compounds in the Treatment of Infections of the Nasal Sinuses, Arch. Otolaryng. **40**:481 (Dec.) 1944. Sulfadiazine in the Prevention of Respiratory Disease, Office of the Surgeon General, Bull. U. S. Army M. Dept., December 1944, no. 83, p. 5.

flowers and weeds; protein from fowl feathers and animal hair, and chemical fumes as tobacco, acids and similar materials.

C. Treatment

1. On duty status
   (*a*) Beverages and fluids limited to water
   (*b*) Desserts avoided
   (*c*) Gas mask to be worn in the plane and on the ground
   (*d*) Ephedrine sulfate solution, 0.5 per cent dropped into the nares as required for nasal ventilation
   (*e*) One capsule (codeine, acetylsalicylic acid and atropine) twice daily, after meals, to reduce irritation and mucus
   (*f*) Attention to morale and psychosomatic factors in the group
   (*g*) Local nasal treatment of a gentle nature may be beneficial
2. In hospital
   (a) Daily dosage of 9 Gm. of sulfadiazine and 18 Gm. of sodium bicarbonate with high intake and output of water is indicated when bacterial rhinitis is present; this may be accentuated or replaced by 100,000 or 200,000 units of penicillin administered parenterally in daily doses for two weeks
   (*b*) Determine sensitivity to allergens by cutaneous tests and elimination diets
   (*c*) Cure the patient's organic diseases
3. Disposition
   (*a*) Return to duty status
   (*b*) Transfer to suitable geographic areas on duty status, compatible with the individual nose
   (*c*) Separation from the service

RESULTS

Fifteen hundred patients were treated for rhinitis (coryza), with and without sinusitis, in the Eye, Ear, Nose and Throat Department during 1944. Less than 5 per cent of the patients required hospitalization. The remaining per cent were treated in the outpatient clinic and continued on a full duty status. Headache and congestion were relieved within twenty-four hours. Conscious obstruction to breathing ceased by the third day. Mucoid and purulent discharge diminished or ceased by the fifth or seventh day. Occasional complications were treated in the hospital. The codeine, acetylsalicylic acid and atropine

regimen in conjunction with sulfonamide drugs and sodium bicarbonate have been used since the days of azosulfamide. It has been satisfactory in private practice and in this part of the country.

CONCLUSION

The highly sensitive mucosa of the upper part of the nasal airway is as susceptible to pressure and changes in temperature as is the more remote periosteal mucosal lining of the sinuses.

The combination of local medication to the mouth of the eustachian tube and daily intramuscular injections of thiamine hydrochloride has been effective in the treatment of eustachian salpingitis, with or without an associated loss of hearing.

A simple procedure is suggested for the relief of most of the nasal signs and symptoms of irritation of the mucous membranes of the nose and eustachian tube.

NEURALGIC SYNDROMES OF THE HEAD

Anatomy and Physiology of Sensory Nerves to the Head

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THE FIRST, second, third, fourth, sixth, eighth, tenth, eleventh and twelfth cranial nerves have no sensory components for pressure, pain, touch or temperature so far as any one knows. This leaves the fifth and the ninth cranial nerve with the intermediary nerve as the only ones capable of carrying painful stimuli from the anterior part of the head to the brain. The sensory patterns of the fifth and ninth cranial nerves are well established and need not be discussed. However, there has always existed some question as to whether or not the intermediary nerve is capable of carrying painful stimuli from the periphery to the brain. The pain about the ear which frequently accompanies Bell's palsy suggests that some element of the facial nerve mechanism may transmit the afferent impulses, since it is pretty well established that the facial and acoustic nerves themselves are entirely free of elements conveying pain, and sensations of temperature, touch and pressure. I have had the experience of cutting the fifth and ninth cranial nerves intracranially and the sensory roots of the first five cervical roots intrathecally and finding that the sensation of touch was still conveyed from the entire side of the face and forehead. Moreover this extensive rhizotomy, which presumably cuts all the pain-carrying roots to the side of the head, did not stop the pain of an aural cancer.

These nerves of the cranium are unique in that they maintain an uncomplicated course from their peripheral endings to the base of the brain and do not enter an entwined plexus of trunks as is observed in the cervical, brachial and lumbosacral plexuses.

Running well up beneath the lower jaw, the base of the tongue, the base of the skull and the posterior part of the head to the vertex is the innervation from the cervical plexus. The filaments of the peripheral branches of these nerves become interwoven in a most complicated fashion before reaching the spinal cord so that no one periph-

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From the Department of Neurosurgery, Jefferson Medical College and Hospital.

Presented before the Section on Laryngology, College of Physicians, Feb. 21, 1945.

eral trunk enters the cord by a single nerve root but enters through all the upper cervical sensory roots of the cervical plexus.

That painful sensations arise in the aforementioned nerves and are transmitted by them to the brain is an undeniable fact supported by daily observation in neurosurgical clinics. Cutting of any of these nerves peripherally or at their sensory roots gives constant absolutely predictable areas of anesthesia.

A second set of nerves over which painful sensory impulses may possibly travel from the head to the brain are those of the autonomic nervous system. The physiologic facts to prove the existence of pathways for pain in this system are so meager as not to be worthy of comment before a group of clinical otolaryngologists with common sense. To prove the existence of such pathways requires the arduous accumulation of isolated theoretic concepts scattered through the literature and a few fortifying clinical observations, which are neither constant nor predictable in terms of a specific surgical procedure resulting in a specific surgical effect.

The third great factor in the causation of pain is the physiologic reaction of the sensory centers of the brain—the thalamus and the cerebral cortical center. That such centers exist is indisputable and that they may hold the undivulged answer to many of the riddles of cephalic pain is a possibility about which physicians know little at the present time.

PAIN SIMULATING NEURALGIA OF THE HEAD

The great majority of pains commonly occurring about the head are readily identified as the result of some local gross pathologic condition or of a demonstrable lesion. Toothache is a typical example. It can be recognized by the local discomfort, the swelling and the discoloration at the location of the inflammation. The same can be said for acute and subacute infections of the sinuses and the ear and diseases of the temporomandibular joint. The roentgenologic examination, the recording of the temperature, the leukocyte count and the sedimentation test give corroborative evidence which makes the diagnosis unmistakable. Indeed, the patient himself most frequently is aware of the general nature of his discomfort. Somewhat more difficult is the diagnosis of cancerous disease of the nose, the throat or the cranial cavity. While cancer of any of these may cause pain in the head, the pain is not usually confused with the chronic shooting, sharp pains of cephalic neuralgias.

An aneurysm at the base of the skull and a tumor on or near the trigeminal nerve may give rise to sharp radiating nerve pattern pains, but there are practically always other tell-tale objective findings to

make the diagnosis positive. Perhaps in isolated instances the oral pain of beriberi may be difficult to diagnose, but beriberi, too, has its general systemic manifestations.

The so-called primary or essential neuralgias about the head which may ramify over many parts of the face or the neck and be without obvious objective signs are far more elusive to the diagnostic clinician. Their recognition is further complicated by the necessity of basing a decision almost entirely on one's interpretation of the patient's own description of his discomfort, which in many instances is colored and inadequate, owing to ignorance, mental apathy, volubility and the lack of any fixed universal terminology to fit human suffering. If this difficulty is aggravated by the patient's inability to understand clearly or to express himself in the examiner's tongue, the classification of the neuralgic ailment enters the category of a guessing contest.

DEFINITION OF THE SUBJECT

In this discussion I shall limit myself so far as practical to the consideration of those pains of the head which are purely neuralgic in type; that is, to those pains occurring in patients in whom no gross lesion in or near the nerve can be demonstrated and in whom there is no objective evidence of nerve disease resulting in a loss of sensation or paralysis of muscles.

MIGRAINE

In terms of paroxysms migraine is probably the most common neuralgia affecting human beings. While it usually produces characteristic symptoms, it may often closely imitate other neuralgias, and indeed may be indistinguishable from them without the most careful and prolonged observation. Most often it conforms to the common description of it as being a hemicrania, but many times the pain assumes the most bizarre pattern of distribution. Typically, it is a familial disease, and the incidence of "sick headache" in other members of the immediate family is of great diagnostic importance. It usually starts early in life and sometimes leaves during adolescence, but most often it becomes more severe with middle age. The onset and the course of the discomfort are exceedingly constant and characteristic. The onset is insidious, and the victim may become aware of a disabling headache without realizing just when it began but knowing well that it started many hours before. It gradually increases in intensity until within several hours it has reached its maximum, to remain constant for some hours, and then it leaves as gradually as it began.

Associated subjective phenomena are common and usually precede the onset of pain. The most common sensation is that of a scintillating scotoma which starts gradually as brilliant spangles or jagged streaks

of bright light clusters. This visual aura grows in size and brilliance until there is almost blindness because of the overshadowing of the visual objects. This gradually disappears to be followed in a period of minutes to an hour or two by the onset of headache. Sensory phenomena such as paresthesias and numbness may occur in the face or the extremities.

The location of the headache is most commonly in one supraorbital region. It usually spreads to the bridge of the nose, the temple and the orbit and may involve both sides of the head. It sometimes extends to the upper jaw, to the back of the neck and into the region of the mastoid process. Nausea and vomiting may occur. The attacks may occur several months apart, or they may recur daily or be repeated so often as to keep the victim suffering with a continual headache for years. Women are more prone to have the attacks just preceding their menses, and the disease has a tendency to subside following the menopausal years in both women and men.

There is usually marked tenderness over the course of the supraorbital nerve at the height of and following the headache. This may be so severe as to prevent combing of the hair or wearing of a hat during the attack.

There are many theories as to the cause of migraine, but little is actually known about its etiologic mechanism. The most that one can say is that it is a combined disturbance of nerve and sensory brain center, of unknown physiologic origin.

The treatment is medical, acetylsalicylic acid being given in mild attacks. A godsend to these sufferers has been ergotamine tartrate given by mouth or by hypodermic injection at the very onset of the headache. Resection of the sensory root of the fifth cranial nerve, peripheral section of the supraorbital nerve, removal or injection of the sympathetic nerves in the neck, ligation of the external carotid artery or removal of the sphenopalatine ganglion do not stop the pain.

TIC DOULOUREUX

Symptoms.—Tic douloureux, or major trigeminal neuralgia, is a disease of the sensory elements of the fifth cranial nerve characterized by "sharp," "shooting," "stabbing," "cutting," "lightning-like" pain in the anatomic distribution of the whole or of one of the branches of this nerve. Each individual sharp pain usually lasts only a fraction of a second, and the pains may occur seconds, minutes or hours apart. When they once start, they are apt to be repeated one after the other, so that the patient receives repeated shocks in rapid succession. In between shocks there is a dull ache which warns the patient that another sharp pain may occur at any moment. During this interval any stimulation of the area covered by the branch affected may start

a paroxysm of pain. This point of sensitivity is often referred to as a "trigger zone." It is most often found just above the eye in supra-orbital neuralgia, on the upper lip in neuralgia of the second branch and at the corner of the mouth or the side of the tongue when the third division is affected. Eating or talking is apt to start the pains, so that the patient will almost starve rather than take food into the mouth. Often he will come to the office with his whole history written out to keep from being drawn into a conversation.

So sudden and sharp are the pains that it is impossible for the patient not to wince when one strikes; naturally, this facial movement takes place directly at the site of the pain, and it is this movement that has often led to the erroneous conclusion that the pain originated from a branch of the facial nerve and therefore the name given this disease—"painful tic."

The pain occurs most often in the lower teeth and almost as frequently in the upper ones. It is much more uncommon in the upper branch. For this reason the teeth are frequently pulled and the sinuses opened in an attempt to effect a cure. Careful observation of the patient in an attack and a true interpretation of the history of the case will disclose a pain that is comparable to none of the pains about the face or the head caused by other diseases of these structures. It is so utterly unlike any other pain about the face that it would seem strange that the diagnosis is so often missed. Once the pain starts, it probably never subsides permanently. I have as yet to see or hear of an authentic case in which the pain has stopped for good without operation. It may leave for days, months or years, but it is sure to return if the patient lives long enough. Strange to say, the remission may follow some surgical procedure, such as the extraction of a tooth or an operation on a sinus.

Anatomy.—For many years there was confusion regarding the difference in function between the fifth cranial (trigeminal, trifacial) and the seventh cranial (facial) nerves. This was largely because it was known anatomically that both of these nerves send branches to the same general topographic parts of the head. Almost every single patient seen by the neurosurgeon, even today, has at some time been told that if he has the fifth cranial nerve cut for relief of the pain he well be disfigured by paralysis of the face for the remainder of his life. Many suffer the atrocious pain of tic douloureux for years because of this fear. Charles Bell in 1821 proved conclusively that all parts of the fifth cranial nerve are separate and distinct from those of the seventh cranial nerve. He first demonstrated this fact on an ass by completely cutting the infraorbital nerve which was known anatomically to be the branch of the fifth cranial nerve supplying the upper lip. Following this procedure, stimulation of the facial nerve caused a

contraction of the muscle in this same upper lip. Prior to this experiment he almost missed the point when he cut both infraorbital nerves and noted that the animal had difficulty in taking food into its mouth. He at first concluded from this that the muscles of the upper lip were paralyzed; later he made the correct observation, on the same animal, that it was unable to grasp hay and grass between the lips because of the anesthesia of the margins of the lips.

Later Bell had occasion to cut the supraorbital nerve in a case of tic douloureux without producing paralysis of the frontalis muscle of the forehead. This proved conclusively to Bell, and the test of time has shown, that although both the fifth and the seventh cranial nerves send branches to the same anatomic locations, their functional activities are entirely different—the fifth supplying sensation to the forehead, the seventh producing motion in the frontalis muscle.

By referring to the anatomic drawing, figure 1, it will be noted that the fifth cranial nerve leaves the pons by two branches—a tiny motor branch, about 1 mm. in diameter, and a larger sensory root, from 3 to 4 mm. in diameter. The sensory root passes into the gasserian ganglion. Leaving the ganglion, which lies on the apex of the petrous portion of the temporal bone, are three large branches entirely sensory in function. The uppermost branch (ophthalmic division) makes its exit from the skull through the superior orbital fissure where it passes beneath the roof of the orbit. It supplies roughly the orbital content and the forehead to the vertex of the scalp.

The middle branch (maxillary division) makes its exit from the skull by way of the foramen rotundum, traversing the pterygopalatine fossa and entering the floor of the orbit. Branches from this location pass to the upper teeth and the lip. In the pterygopalatine fossa it gives off tiny branches to the sphenopalatine ganglion, which in turn sends branches to the mucous membrane of the nasal cavity and the roof of the mouth.

The third branch (mandibular division) leaves the skull through the foramen ovale. It passes to the region of the lower jaw, supplying the entire mandible, the skin and the mucous membrane outside and inside this structure and the anterior two thirds of the tongue.

The anterior (motor) branch passes behind but not through the gasserian ganglion and leaves the skull along with the third or mandibular division. Outside the skull it divides into branches which supply the muscles of mastication (internal and external pterygoid muscles, temporal and masseter muscles). All of these muscles are concerned with approximation of the teeth and have nothing to do with the movements of facial expression. To cut this branch will in no way disfigure the face. If the branch of one side only is cut, the mouth will still close almost perfectly.

Causes.—Little is known about the cause of tic douloureux. The pathologic process is seldom demonstrable, and the correction of disease in the area of distribution of the pain never cures the patient of the pain. Operations on sinuses, pulling of teeth and correction of other pathologic processes peripheral to the ganglion are notoriously ineffective in relieving the pain. It would appear as though the process which

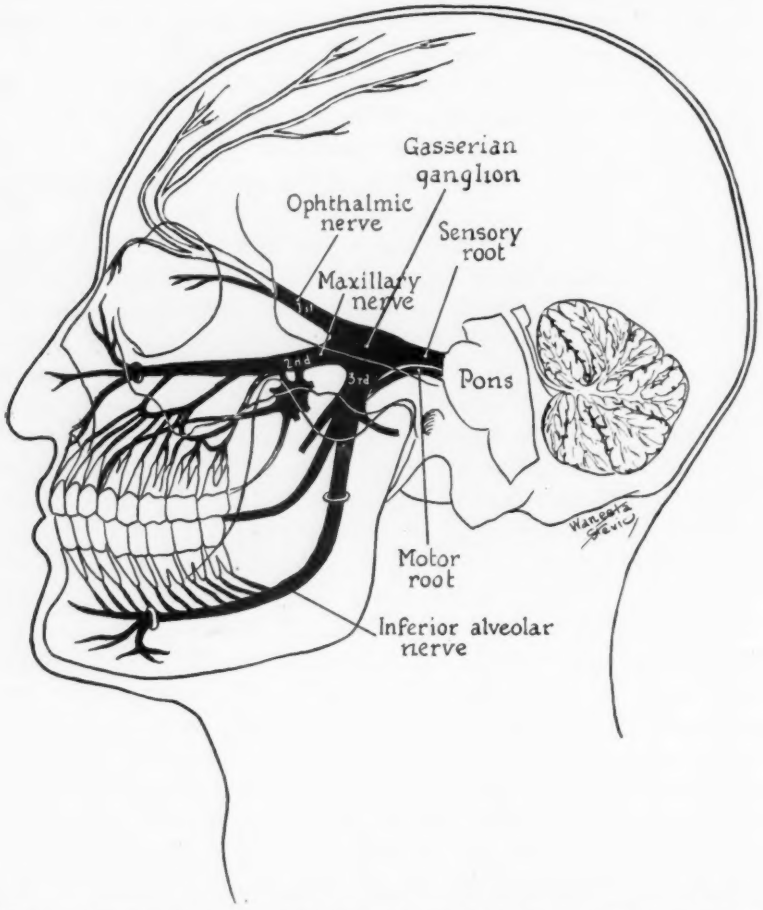

Fig. 1.—Drawing to show how the fifth cranial nerve springs from the pons and how its sensory branches are distributed to various parts of the head. For the surgical treatment of tic douloureux, only the lower one half of the sensory root is sectioned between the pons and the gasserian ganglion.

is causing the neuralgia exists either in the gasserian ganglion or between the ganglion and the origin of the nerve in the brain stem. Occasionally the surgeon finds a definite pathologic change which unquestionably is the cause of the typical tic douloureux. On one

occasion a tumor of the gasserian ganglion was uncovered at operation. On a second occasion a large, tortuous sclerotic basilar artery was so looped laterally as to press tightly against the sensory root of the fifth cranial nerve. On a third occasion a large, pearly body tumor was found tightly compressing the sensory and motor roots of the fifth cranial nerve. In the last instance the patient had been given relief by alcohol injected into the infraorbital foramen, for pain in that distribution, for a period of two years. One year after the onset the pain began in the third division; this was likewise relieved by injected alcohol for a period of a year. When the pain recurred in the second division, the patient returned for operation, and a tumor was found. There was no suspicion of its presence prior to the exploration, which was carried out beneath the cerebellar hemisphere.

Treatment.—Drugs are notoriously ineffective in giving even temporary relief. The pain occurs so unexpectedly and is so severe that even large doses of morphine give no comfort. Although this outrageous pain lasts over many years, one never sees a person afflicted with the disease addicted to the use of narcotics.

1. Trichloroethylene. This drug has been used for a number of years for the purpose of stopping the attacks of pain. It is sold in an ampule containing approximately twenty drops, which can be broken in a handkerchief. Being volatile, it may be inhaled with several deep breaths. It acts specifically on the fifth cranial nerve, producing anesthesia over its distribution so that a numbness is noted about the face over the distribution of the fifth cranial nerve. Caution must be used not to hold the drug too close to the nose while it is being inhaled, as it is apt to irritate the tip of the nose. It should not be used more than four or five times a day. While it does no harm to try this drug, for apparently it is harmless used in the way described, at the same time it has proved entirely unreliable in the hands of most neurosurgeons.

2. Injection of alcohol. Alcohol injected into the peripheral nerve trunk affected by this disease will frequently give relief for as long as three years. The success of this procedure depends on depositing the alcohol exactly in the substance of the nerve trunk. From 1 to 2 cc. of alcohol is sufficient to kill the nerve. It is injected into the first or ophthalmic branch where it comes out of the orbit just above the superciliary ridge. At this point it usually divides into three small branches. The infratrochlear branch comes out just beneath the pulley-like loop of the superior oblique muscle (1 cm. directly above the inner canthus of the eye) and passes down the side of the nose. Another branch, the supratrochlear, comes from just above the pulley and about 2 cm. from the midline. This supplies the mesial part of the forehead. The

third branch, the supraorbital, is the largest one of the three and emerges in the supraorbital notch, which can usually be palpated with the finger and located from 2.5 to 3 cm. from the midline. Sometimes this notch is bridged over by bone, and then there is a foramen through the superciliary ridge. This nerve supplies the greater part of the forehead and is usually the one affected in pain above the eye.

The superior maxillary division should be blocked by placing the needle in the ptergyopalatine fossa between the foramen rotundum and the exit of the nerve in the roof plate of the antrum. The needle should be placed into this location either by going just above the zygoma and passing perpendicularly to this space or by placing the needle just below the zygoma and about an inch and a half (3 cm.) in front of the external auditory meatus. Alcohol injected in this location should anesthetize the roof of the mouth, the upper teeth and the upper lip.

Injection into the third or mandibular branch of the fifth cranial nerve should be made in one of two locations. The most common site for the injection is just below the zygomatic arch about 2 cm. and 3 cm. anterior to the external auditory meatus. The needle is passed upward and inward to the foramen ovale. At this point both the motor branch to the muscles of mastication and the sensory branch which supplies the tongue and the lower jaw will be caught. When the pain is restricted to the lower teeth an injection may be attempted into the inferior alveolar nerve at the point where it enters the mandible. This procedure is carried out exactly as in performing a block of the inferior alveolar nerve with a local anesthetic. If the pain also involves the side of the tongue, it is much better to block the nerve at the foramen ovale than to try to produce good anesthesia of the lingual nerve, which is difficult because of the location of the nerve in the soft structures of the mouth. Whenever possible, the injection should be performed with the patient under nitrous oxide or pentothal sodium anesthesia. If procaine hydrochloride is injected as a preliminary, it is apt to dilute the alcohol and make the latter less effective. Without anesthesia of any kind injection into the nerve is a very painful procedure.

If the pain is restricted to the upper teeth and the upper lip, the alcohol may be injected into the infraorbital foramen just beneath the orbit.

The injection of alcohol should be performed in every patient who is elderly and who is necessarily a poor operative risk, and in any patient whose life expectancy is shorter than the length of time the nerve will require to regenerate. It may also be performed in a young person as a procedure preliminary to later operation. It does not seem wise to inject alcohol repeatedly for tic douloureux in a young person with a long life expectancy because of the mental horror associated with the knowledge that the pain will certainly return. The procedure of inject-

ing alcohol into the gasserian ganglion by passing the needle through the foramen ovale is a dangerous one and is to be recommended only in exceptional instances. When the injection is made into the ganglion the alcohol must enter the subarachnoid space. This space communicates with all the internal structures of the brain. For this reason injection into the gasserian ganglion is hazardous.

3. Operation. There are two standardized operative procedures directed toward the cutting of the whole or a part of the sensory root of the fifth cranial nerve between the ganglion and the brain. When sectioned in this location, the nerve will not regenerate. The first approach is by way of the temporal fossa, first performed by Victor Horsley and later improved by Frazier and Spiller. The second operative procedure is that developed by Dr. Walter Dandy, the one which I now use entirely. The cutaneous incision is made just back of the mastoid process and directly over the cerebellar hemisphere. The cerebellum is raised and the basal nerves exposed. By passing directly above the seventh and eighth cranial nerves the fifth nerve is uncovered. By this approach the sensory root is either partially or totally cut, depending on the desires of the surgeon (fig. 2). It is possible by cutting approximately one half of the lower part of this root to relieve the sufferer of the sharp stabbing pains and yet retain touch, temperature and pain sensation over the greater part of the area of distribution of this nerve. The motor branch of the fifth nerve is less likely to be injured or cut by this procedure, and certainly the retention of all types of sensation in the face is a desirable feature of this operation. Technically it is an easier procedure for those skilled in intracranial surgery to carry out than the approach beneath the temporal lobe of the brain.

Operation is the only permanent cure for this disease, and the mortality from the procedure is so low that in the majority of instances it is to be recommended. The only possible bad effects are those incident to the complications resulting from corneal anesthesia in cases in which the entire nerve root is cut. This occurs rarely and, if it is properly handled by temporarily suturing the lids together, is not to be considered an objection to the operation. Facial paralysis occasionally occurs from edema arising in the petrous portion of the temporal bone, which strangulates the facial nerve as the nerve passes through the facial canal. It comes on several days to three weeks after the operation, is seldom complete and always disappears entirely after three or four months' time. It has never occurred in my patients operated on by the posterior approach.

GLOSSOPHARYNGEAL NEURALGIA

Glossopharyngeal neuralgia is similar in character to tic douloureux, and its recognition is equally important. It differs from tic douloureux

only in its area of involvement. Since the ninth cranial nerve sends sensory fibers to the base of the tongue, the soft palate, the tonsillar fossa and the internal ear, the pain of this disorder is distributed to

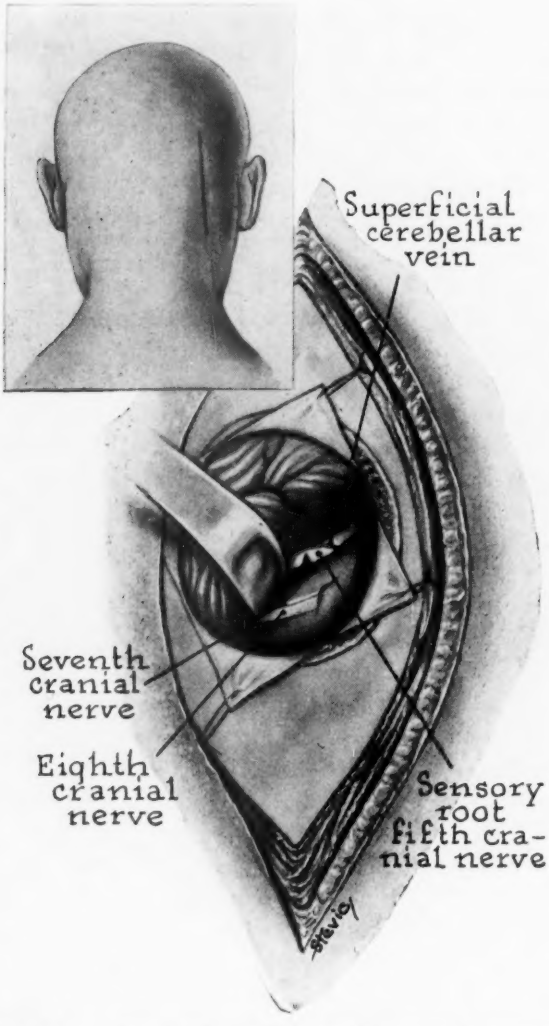

Fig. 2.—Drawing to show how the fifth cranial nerve is exposed for partial section by lifting the cerebellar hemisphere in the surgical treatment of tic douloureux.

these areas. There often is extreme pain on talking and particularly on swallowing, because food must pass over the hypersensitive zone in the throat.

Reichert[1] called attention to neuralgia of the tympanic branch of the glossopharyngeal nerve (nerve of Jacobson), characterized by sudden, brief paroxysms of pain deep in the ear and without a "trigger zone" in the throat. Sometimes the discomfort is referred to the neck directly below the ear at the angle of the jaw. While this is a much rarer disease than tic douloureux, its recognition is important because of the intense suffering attending it and the necessity for specific treatment.

A cure of this disease can be affected only by intracranial section of the ninth cranial nerve through the posterior fossa. This nerve lies superficially, is easily recognized and can be completely cut. Anesthesia of the throat and the tongue over the small area supplied by the nerve is of no concern. The injection of alcohol is impossible because of the deep anatomic position of the nerve. Medical treatment is as ineffective as it is in tic douloureux.

ERYTHROMELALGIA (HISTAMINIC CEPHALGIA)

Horton[2] gave credit to Perry (1789) for first discovering and fully describing this neuralgic syndrome, which has now been firmly established by many subsequent observations of numerous clinicians. Furthermore, he has been able to control the disease in a great percentage of cases by the simple immunization of the sufferer with histamine phosphate.

In the words of Horton:

> Histaminic cephalgia is characterized by a unilateral headache, which usually begins in the later decades of life, is of short duration as it generally lasts less than an hour, commences and often terminates suddenly, tends to awaken the patient at night one to two hours after he has gone to sleep and is frequently eased by the patient sitting up or by standing erect. It is associated with profuse watering and congestion of the eye, rhinorrhea or stuffiness of the nostril, increased surface temperature and, often, swelling of the temporal vessels of the involved side of the head.
>
> Pain is the outstanding complaint. It is constant, excruciating, burning and boring; it involves the eye, the temple, the neck and often the face. So severe and frequent are the attacks of pain (as often as twenty times a week) that practically every patient had contemplated suicide. The pain is not confined to the distribution of any cranial nerve but has a tendency to conform to the ramifications of the external carotid artery. During and following the attacks, marked tenderness is usually found when pressure is applied over the branches of the external and common carotid arteries. In the first stages of an

1. Reichert, F. L.: Tympanic Plexus Neuralgia: True Tic Douloureux of the Ear or So-Called Geniculate Ganglion Neuralgia; Cure Effected by Intracranial Section of the Glossopharyngeal Nerve, J. A. M. A. **100**:1744 (June 3) 1933.

2. Horton, B. T.: The Use of Histamine in the Treatment of Specific Types of Headache, J. A. M. A. **116**:377 (Feb. 1) 1941.

attack, compression of the common carotid artery and sometimes the temporal artery frequently gives prompt relief. A few patients have discovered for themselves that compression of the artery on the side of the pain gives relief.

The pain is likely to occur suddenly at night and to be aggravated by alcoholic beverages. The treatment of desensitizing the patient with histamine phosphate is carried out with daily subcutaneous injection of a solution of this substance. The solution comes in 1 cc. ampules which contain 0.275 mg. of histamine phosphate. The first dose is 0.25 cc. The daily dose is gradually increased until by the twentieth day 1 cc. is given. The gradual increase of the dose continues until the headache stops, then a maintenance dose is given one to three times a week. Overdosage is to be avoided, as this seems to cause the headache.

Horton should be given full credit for his contribution to the solution of the problems of this syndrome. Its clinical recognition would help reduce the number of unexplained facial neuralgias.

POSTHERPETIC NEURALGIA

Persistent neuralgic pain following the healing of a herpetic eruption is a well established clinical entity. It may affect any location on the head and usually follows a rather extensive cutaneous eruption and not the herpes simplex type. Most patients show extensive healed scars over the general area of the distribution of the pain and give a definite history of having had acute lesions with considerable distress at the onset of the disease, which has persisted.

The pain is described as being a burning, itching, aching soreness and is seldom sharp or shooting. It is constant through each twenty-four hours of the day and aggravated by exhaustion and nervousness. There is sometimes hypesthesia or hyperesthesia over the affected area. Once it persists longer than three or four months, it becomes so thoroughly fixed that it probably never entirely leaves. When it affects the region about the mouth, there are often bizarre facial movements with smacking of the lips.

There is no treatment that will cure this disorder. Section of the nerves to the affected part with production of complete anesthesia will not cure it. Usually the patient will state that the pain is entirely gone immediately following a surgical procedure, but in three or four weeks it is back as bad or worse than before. Even chorodotomy, when the pain is in the lower extremities, has failed to help these patients, in my experience.

One wonders if this pain does not reside in the brain and if it may not be psychogenic—at least in its chronic form.

POSTOCCIPITAL NEURALGIA

Chronic neuralgia of the nerves to the occiput is common in aged persons. It is not often constant, but it persistently recurs lasting for days at a time. There is seldom tenderness, and no loss of cutaneous sensation. The pain is described as an aching soreness without sharp shooting pains. It has the character of a genuine inflammatory neuritis and is probably caused by inflammatory changes about the vertebral foramens in the bony and ligamentous structures of the cervical portion of the spine.

Removal of the nerves seldom helps the pain, but this should be tried when it is restricted to a definite superficial nerve branch. Injection of alcohol does no good and may even make it worse. Injections of procaine hydrochloride on several occasions seem to have helped. Rhizotomy of the first four cervical sensory roots will stop the pain but is rather drastic for most of these patients.

Most of them can be kept comfortable with acetylsalicylic acid, codeine and heat, or by fixing the neck in a well fitted leather collar for a few weeks.

SYPHILITIC NEURALGIA

Syphilis, when it inflames the sensory roots of the cranial nerves, may cause dull aching and sharp shooting pains of the head. These are usually worse at night but may occur during the day. Sometimes they are so acute as to imitate the pains of tic douloureux.

A careful neurologic examination usually reveals other signs of syphilis of the nervous system, such as Argyll Robertson pupils and absence of the patellar reflexes, but many times the pains are the only indication.

When sharp facial pains do not fit the symptom complex of a well known disorder, the spinal fluid and the blood should be examined for syphilis.

POST-TRAUMATIC HEADACHE

Headache following more often trivial than violent injury of the head is a common occurrence. There may be a fracture of the skull, but usually not. The pain may involve the whole top of the head or be in the frontal or the occipital region, or both, and it is usually constant and aggravated by fatigue and nervousness. Chronic slight dizziness many times accompanies it.

This condition must not be confused with the headache of subdural hematoma following injury of the head, which is likely to be accompanied by stupor.

Encephalography fortified by psychotherapy is probably the best treatment. This procedure also rules out a possible hematoma. Ligation of the middle meningeal arteries has produced cures and is to be recommended if the pain is persistent and severe.

AURICULOTEMPORAL NEURALGIA

Some time ago I reported [3] 2 cases of pain in the auriculotemporal nerve, with sharp pains shooting over its distribution to the region above the ear on motion of the lower jaw. Recently I have seen it in a more aggravated form, the ache being constant and keeping the patient awake at night. Talking and eating were impossible without great distress, and the skin over the temple to the vertex was exquisitely tender.

Resection of the auriculotemporal nerve cured the first 2 patients; the third was apparently cured by fixing the teeth together.

TEMPORAL ARTERITIS

This is a disease of elderly persons which is characterized by marked tenderness over the temporal artery, malaise, fever, general weakness, local cephalic pain and redness, and mild leukocytosis. The underlying pathologic process is an acute inflammation of the temporal artery. The pain can be stopped by removing the artery.

NEURALGIAS OF NASAL ORIGIN

Chronic neuralgias of the head that arise as a result of abnormal intranasal anatomic relations and pathologic developments are a common occurrence in the practice of any rhinologist. One readily learns to recognize these throbbing pains recurring about the root of the nose, in the forehead, the eye and the upper jaw. Direct inspection discloses a deviated or thickened septum, polypi, an enlarged turbinate against the septum or other abnormality. Shrinking or cocainization of the mucous membrane stops the pain, and an appropriate operation cures it permanently. This type of pain is encountered and cured by the rhinologist so frequently as to need little comment from a neurosurgeon. However, pains in the structures mentioned and which definitely appear to be nasal in character are many times resistant to cure. The discomfort continues even after multiple meticulous surgical procedures have long since eradicated any possible gross pathologic process. These are the patients who return to the rhinologist week after week, month after month and year after year for intranasal packing, sprays, local applications. They are addicted to the use of nasal drops and nasal inhalers.

In a small group of these patients the pain must be psychogenic in origin. Other widespread complaints without obvious pathologic origin and aggravated by nervousness and exhaustion point to the

3. Jaeger, J. R.: J. Colorado Dent. Soc., December 1940, p. 1.

4. Paviot, J.; Chevallier, R.; Guichard, A., and Damez, M.: Craniofacial Sympathalgia of Migraine Type with Dilatation of Temporal Arteries in Course of Isolated Cardiac Rheumatism, Lyon méd. **154**:45 (July 15) 1934.

probability of a functional disorder. The chance of effecting a cure by surgical treatment is hopeless, and operation aggravates the mental fixation. Psychotherapy is the only hope for these persons and it usually has to be applied for a lifetime. One wonders if repeated applications to the nose combined with kind words from a rhinologist with a personality that inspires confidence and contributes assurance is not fully as good as psychiatric treatment, even though less specifically scientific.

A larger group of patients with chronic nasal pain and without objective disease to account for it unquestionably suffer from chronic inflammation of the nerves supplying the nasal structures. Perhaps rhinologists do not sufficiently appreciate the extreme sensitivity of the nasal mucous membrane. One may readily manipulate, rub or otherwise stimulate the body's skin or the mucous membranes of the alimentary or the genitourinary tract without great discomfort, but let a small piece of cotton or a few molecules of irritating gas strike the respiratory system and there is an instantaneous violent protective reaction. The nerves to the upper respiratory tract are the most irritable in the human body and for this reason are probably the most susceptible to pain when affected by inflammation. Weir Mitchell first described the chronic pains, trophic disorders and muscular incapacity when neuritis affects nerves of the extremities. He termed the disorder "causalgia" and emphasized its chronicity and resistance to attempted cures. Surgical exploraton, massage and other local treatment to the affected nerve only make the condition worse. Surely there is reason to believe that a similar incurable neuritis affects the nasal nerves.

To Greenfield Sluder probably should go the credit for emphasizing the nature of chronic inflammatory disease of the nasal nerves. His name seems to be more commonly associated with massive neuritis of the sphenopalatine ganglion. Since this nerve center innervates the greater part of the intranasal mucosa and also carries the major part of its autonomic innervation, any neuritis affecting it causes widespread nasal signs and symptoms. It is only natural that Sluder should have emphasized the relation of infection of the sphenoid sinus to sphenopalatine neuralgia when one realizes the close approximation of these two structures.

Sluder's [5] description of his neuralgic syndrome caused by irritation of the sphenopalatine ganglion will bear repetition:

> Lower Half Headache—The Neuralgic Syndrome with the Usual Forerunner. A patient presenting all the features will tell of a coryza of lesser or greater severity—sometimes astonishingly slight and often forgotten, or it may have produced a post-ethmoidal-sphenoidal empyema. [It has been my observation that many times patients are unconscious of coryzas, but still examination shows

5. Sluder, G.: Headaches and Eye Disorders of Nasal Origin, St. Louis, C. V. Mosby Company, 1918, pp. 69-70.

that an inflamed membrane is the cause of headache, usually sphenoidal.] A short time later, pain began at the root of the nose, in and about the eye, the upper jaw and teeth, sometimes also in the lower jaw and teeth, and extended backward to the temple and about the zygoma to the ear, causing earache; emphasized at the mastoid, but always severest at a point 5 cm. back of that [which is almost always quite sensitive to pressure]; thence reaching backward by way of the occiput and neck, it may extend to the shoulder blade and shoulder (less often to the axilla and breast), and in severe attacks to the arm, forearm, hand and even the finger tips. This is the most frequent picture, but at times there may be also a sense of "stiff" or "aching" throat; or of pain (oftener itching) in the hard palate; or pain inside the nasal fossa; or a feeling that the teeth are "too long"; or a perverse (metallic) sense of taste (parageusia) (rare) or scotoma scintillans (rare) or salivation (rare). Itching of the shoulders and upper extremity has been observed. The sense of taste is slightly less acute over the anterior two thirds of the tongue on the affected side. Rarely, in the beginning or at the height of the attack it is slightly more acute. The arch of the soft palate is higher on the affected side; the uvula and the dimple which forms above it on gagging are deflected to the well side. A slight blunting of sensation is found in the nose and throat as far down as the tonsil on the affected side. There may be a sense of stuffy ears, which are easily inflated with but short relief.

In his description Sluder supplemented the pure sensory disturbance with phenomena which he attributed to irritation of the autonomic nervous system so that lacrimation, dilatation of the pupil, sneezing, rhinorrhea and congestion of the conjunctiva were added to the symptoms of pain.

While one might well disagree with Sluder on many details of his published ideas, yet fundamentally neurologists at least will accept his explanation of the distribution and the character of the pain caused by disease of the great center of the sensory supply to the nose.

When it comes to the neurosurgical cure of chronic neuralgia which is characteristically of nasal origin in a patient in whom all nasal pathologic processes and deformity have been corrected one has a real problem on one's hands. In the words of Sluder, "frequently in severe cases that have been relieved by injection of alcohol, the benefit proved so transitory as to arouse the inquiry: 'Was it worth while?'" However, when the surgeon has eradicated all intranasal pathologic processes that might resonably be expected to cause pain of nasal origin, the problem should be approached from the standpoint of an inflammation of one of the branches of the fifth cranial nerve. Obviously the nerves involved must be those of the first and second branches of the trigeminal nerve.

Nasal neuralgias, while persistent, are usually not severe enough to warrant resection of the sensory root of the trigeminal nerve. So far as I know, there has been practically nothing published on this

approach to the problem. Then, section of the sensory root would not get rid of the autonomic connection by way of the sphenopalatine ganglion.

Practically the entire specific surgical attack has been on the sphenopalatine ganglion and that through injection of alcohol. Injecting alcohol into the sphenopalatine ganglion in a satisfactory manner is an exceedingly difficult procedure and requires years of experience, profound judgment and extreme patience on the part of the operator. It should be done in the hospital with the patient under sedation and general anesthesia and with the understanding that several attempts may be necessary to get the right result. It makes little difference which approach to the ganglion is used so long as one realizes the dangers involved, which are largely concerned with injury to the ocular structures. Blindness and double vision have occurred many times following attempts to make an injection into this ganglion. The only reliable objective indication of a completely successful alcohol block of the sphenopalatine ganglion and its branches is total anesthesia of the upper jaw, which should persist for at least one year.

In some cases it may be advisable to remove the ganglion surgically, especially in those instances in which alcohol has given relief for some months or years and a more prolonged effect is desired, or in which the injection of alcohol has not been successful. The approach is through the anterior and posterior walls of the antrum and beneath the lip. In the sphenopalatine fossa are many branches of the internal maxillary artery, bleeding from which must be controlled by electrocoagulation and suction. Three patients on whom I performed this operation have been completely relieved for many years.

I am sure that a more careful study of chronic nasal pains from the standpoint of specific nerve structures affected will result in many cures heretofore considered unattainable.

BUCCAL NEURALGIA

Reichert [6] attempted to segregate a small group of cases of atypical facial neuralgia from this large group of cases of ill described facial pain or pains and called it "buccal neuralgia". He defined this as being:

> . . . a form of atypical . . . neuralgia, characterized by a burning, boring, aching, throbbing, toothachy pain in the region of the lip, cheek, gum, tongue, maxilla, nose, upper jaw and sometimes lower jaw, spreading at times to the zygoma, into or behind the eyeball or to the temporal region. It may be present for minutes to hours to days, with exacerbations of sharp, stabbing pain. Light touch to the involved part rarely starts an attack, but chewing, talking, local

6. Reichert, F. L.: Buccal Neuralgia: A Form of Atypical Facial Neuralgia of Sympathetic Origin, Arch. Surg. **41**:473 (Aug.) 1940.

pressure or cold frequently induces an exacerbation. Hypersensitiveness to pressure over the carotid, facial and temporal vessels is frequently elicited.

He expressed the belief that the pain travels by way of the sympathetic nerves along the facial artery and veins and implied that the disorder is a neuralgia of these nerves. He reports the treatment of 30 patients with the symptoms mentioned. Seventeen were treated by resecting the facial artery and vein and their sympathetic fibers at the lower border of the mandible with 13 cures. Eight were treated by an injection of alcohol interrupting the cervicothoracic portion of the sympathetic chain, and 5 were cured. Three who exhibited hypothyroidism were cured with thyroid extract and vitamin B. One patient was relieved spontaneously. One patient was cured by ligation of the angular vein.

It is of interest to note that 10 per cent of these patients also had tic douloureux.

There have been no additional reports in the literature on this disorder so far as I can determine to date. In my own experience I thought I had selected 3 typical cases of the group described by Reichert for this treatment. In all 3 the treatment failed to produce relief. It remains to be seen whether or not this syndrome withstands the test of time.

PSYCHOGENIC NEURALGIAS

The patient with cephalic pains of mental origin vary from the nasal drop, nasal inhaler addicts to the profoundly psychoneurotic persons, many of whom become addicted to drugs and must be institutionalized. The milder psychogenic pains may be almost indistinguishable from genuine organic neuralgias. There are certain tell-tale differences however. Psychogenic pains are usually itching, burning and boring, and deeply seated. They persist day after day and are constant during each twenty-four hours. Under emotional stress and fatigue they are worse. The patients have a generally nervous agitated disposition and may weep readily. They frequently have a multitude of other bodily complaints. They recite at length and vividly the details of every therapeutic procedure that has been used on them, often fortifying this information with reams of writing and records narrating their past symptoms and treatments in meticulous detail.

The most convincing evidence that a pain is psychogenic is its dissimilarity when it is compared with any well established clinical syndrome. Woe be unto the surgeon who even suggests to the patient that he has an operative cure for such a disorder. Once encouraged they cannot be easily shaken off, and if an operation is performed, it becomes a fixed part of the patient's imaginative, highly colored, horrible account to be spread far and wide by a tongue that seems to be hinged in the middle and wagging at both ends. A psychiatrist is the only hope and the surgeon's haven of refuge from such a patient.

FRACTURES OF THE MALAR-ZYGOMATIC COMPOUND

Treatment by Improved Methods and Myoplasty

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FRACTURE of bones of the zygomatic compound is usually accomplished by severe external force. The zygomatic arch is fractured singly more often than the malar bone, 10 to 1. It is therefore understood that the force necessary to fracture the zygomatic arch is less than that required for a similar injury to the malar bone. Compound fracture of the zygomatic arch, except by a weapon, is unknown (Coakley [1]). Fracture of the zygomatic compound is usually caused by direct force, such as that encountered in an automobile injury or a blow by fist or instrument. If the maxillary antrum is thin walled, a blow may drive the malar bone directly into it. Fractures of the zygomatic arch are usually multiple (Coakley and White [2]).

Duverney, in 1751, first mentioned treating this type of fracture. It is believed that he reduced the fracture by intraoral manipulation. Hamilton, in Buffalo, was the next person to mention the treatment of this condition in the literature. He stated that Duverney's method could be used only when the fracture is near the osseous union of the zygomatic arch and the malar bone. Until 1922 not more than a dozen cases were reported of fractures of the malar-zygomatic compound.

SYMPTOMS AND SIGNS

The symptoms and signs of a fracture of the malar-zygomatic compound are: (1) pain; (2) periorbital swelling and depression or flatness of the face; (3) pain on mastication; (4) trismus if the arch is involved, and limitation of the motion of the jaw because of depression of the zygomatic arch against the coronoid process of the mandible (Smith [3]); (5) intranasal hemorrhage on the injured side; (6) diplopia, a most

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From the Brooke Hospital Center.

Read before the College of Physicians of Philadelphia, Section on Otolaryngology, and the Philadelphia Laryngological Society on March 15, 1944.

1. Coakley, W. F.: Personal communication to the authors.

2. Coakley, W. F., and White, M. F.: Fractures of the Zygoma: Seventy-Two Consecutive Cases, Surg., Gynec. & Obst. **77**:360 (Oct.) 1943.

3. Smith, F., in Manual of Standard Practice of Plastic and Maxillo-Facial Surgery, Philadelphia, W. B. Saunders Company, 1942.

important subjective symptom; (7) emphysema from fracture of the antrum and puncture of the antral lining; (8) when the malar fracture extends into the orbit, a resulting depression which can be palpated even in the presence of swelling; (9) subconjunctival hemorrhage, and (10) sensory changes from paresthesia to anesthesia in the region of the infraorbital nerve.

There are four sites of fracture which correspond to the articulations: (1) the frontal process—the superior external orbital margin; (2) the orbital process—the region of the infraorbital foramen; (3) the zygomatic arch, and (4) the zygomatic process of the maxilla (Smith [3]).

Fracture of the zygomatic arch is frequently missed by inexperienced surgeons. Limitation of motion of the mandible is the most constant sign of this condition. Displaced fragments are frequently palpated within and outside the mouth before too much edema is present. Limitation of mandibular motion in the presence of injury should always indicate roentgenologic examination. The verticosubmental or Watters projection will bring out more information in regard to fracture of the zygomatic arch than will any other measure.

TREATMENT

The method and the time of reduction depend a great deal on the physical condition of the patient and on whether a compound or a simple fracture is present. The reduction of the zygomatic arch is a simpler procedure than the correction of a malar fracture.

The method of Matas,[4] 1896, by external approach, is one of the oldest methods and is still very popular. It consists of passing a curved cutting needle from below upward around the zygomatic arch, with a heavy suture, in order to pull through a silver wire. The wire may be twisted over a padded splint, so as to hold the fragment in position. This method is best used for depressed fractures of the zygomatic arch.

Keen [5] described an intraoral approach by a small incision in the buccal fold over the upper second molar and the insertion of a blunt instrument under the zygoma for manipulation and elevation. The method of intraoral incision is not universally favored, although at the Brooke Hospital Center several score of these arches have been reduced by an improved method. After reduction of the fragments, the incision cavity is packed with BIPP gauze (bismuth subnitrate iodoform paraffin paste), a lubricated dressing, and left in position for several days.

4. Matas, R.: Fracture of the Zygomatic Arch. New Orleans M. & S. J. **49**:139 (Sept.) 1896-1897.

5. Keen, W. W.: Surgery: Its Principles and Practice, Philadelphia, W. B. Saunders Company, 1907, vol. 2, p. 146.

The gauze packing also holds the fragments in position. Good results have been obtained with no postoperative infections to date (Wade [6]).

The method of Manwaring-Gill [7] is a method of external approach and is very simple. It consists of grasping the zygomatic fragment with a medium-sized towel clip or with a heavier cow horn dental forceps and of manipulating the fragments into position. Manwaring-Gill in early cases advocated the use of the towel clip method to rock the infraorbital crest into position. This can be accomplished by gloved fingers, sliding the skin of the face over the orbital rim. This pushes the delicate orbital skin out of the operative field. The orbital segment must be strong and well attached to the malar bone to produce traction.

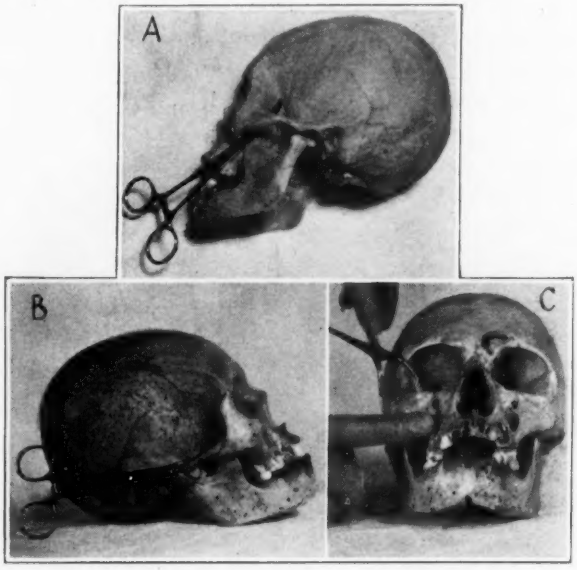

Fig. 1.—Reduction of a fracture of the malar-zygomatic compound. *A*, internal method of Keen. *B*, Manwaring-Gill method. *C*, modification of Manwaring-Gill method.

The Gillies method,[8] or temporal approach, is the method of choice, employed by most experienced maxillofacial surgeons. This procedure can usually be accomplished by local anesthesia, although in private practice intravenous anesthesia is more desirable. A horizontal incision

6. Wade, W.: Personal communication to the authors.

7. Manwaring, J. G. R.: Replacing Depressed Fractures of the Malar Bone, J. A. M. A. **60**:278 (Jan. 25) 1913. Gill, W. D.: Fractures of the Facial Bones with Special Reference to Involvement of the Paranasal Sinuses and Orbits, South. M. J. **27**:197 (March) 1934.

8. Gillies, H. D.; Kilner, T. P., and Stone, D.: Fractures of Malar-Zygomatic Compound, with Description of New X-Ray Position, Brit. J. Surg. **14**:651 (April) 1927.

is made over the temporal muscle in the shaved scalp above and anterior to the ear. A second incision is made in the fascia of the temporal muscle. A long, specially curved binangle elevator is passed between the temporal fascia and the temporal muscle, under the zygomatic bone. The skull is padded and used as a fulcrum in manipulating the fragments into position. Some fractures of the malar bone can be reduced by this method, and over 95 per cent of the fractures of the arch can be repaired by this method. Coakley and White,[2] of the Kings County group, have used the Gillies method in 72 consecutive cases without infection. There have been no reports in the literature of postoperative infection following the use of the temporal approach.

New [9] reported the use of a sharp hook to elevate the arch fragment by passing the hook through the skin and under the fragment.

The treatment of malar fractures usually demands special planning. Manipulation is more difficult, and fixation and traction are frequently

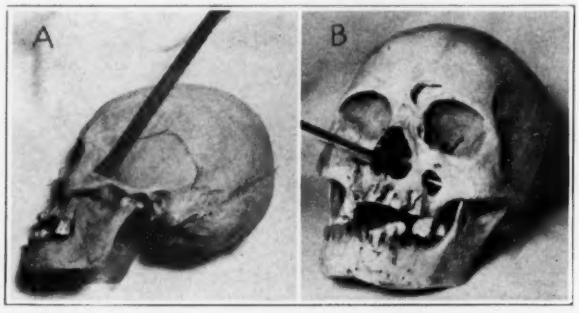

Fig. 2.—Further methods of reducing a fracture of the malar-zygomatic compound. *A*, the Gillies or external approach. *B*, the intranasal method.

required. The fragments are often impacted, and reduction cannot usually be delayed as long as with a fracture of the zygomatic arch.

After a small incision over the malar prominence, Roberts [10] uses a corkscrew-like instrument for elevation and manipulation.

When the malar bone is detached and movable and the fracture extends into the orbital rim, O'Brien,[11] with the patient under general anesthesia, inserts a blunt instrument intraorally under the malar prominence and with a gloved thumb inserted in the conjunctival cul-de-sac rocks the fragment into its proper place.

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9. New, G. B.: Fractures of Nasal and Malar Bones, S. Clin. North America **15**:1241 (Oct.) 1935.

10. Roberts, S. E.: Fracture of the Malar Zygomatic Arch, Ann. Otol., Rhin. & Laryng. **37**:826 (Sept.) 1928.

11. O'Brien, G.: Personal communication to the authors.

Fixation of the malar bone can be accomplished in certain cases by direct wiring with tantalum wire. When traction is needed with external fixation, a plaster of Paris head cap is constructed, and by the method of Ivy and Curtis [12] a screw eye is inserted through an external stab incision in the malar fragment, which is held in position for about a week.

Lothrop,[13] in 1906, was first to use the method of antral approach to elevate the malar bone. An incision is made over the canine fossa into the soft tissues. The detached bone fragments and clots are removed; and a heavy instrument is used to lift the fragments into position. Drainage into the nose should be established by a nasoantral window under the inferior turbinate bone. A packing of the antrum with iodoform gauze helps to hold the fragments in proper position. These packs may be left in position five to seven days, although Smith [3] stated his belief that these packings should not be left in the antrum over forty-eight hours.

Associated fractures of the maxilla can be reduced without a cumbersome plaster head cap, by internal fixation by wiring, as described recently by Adams.[14] A small incision is made over the ridge of the frontal bone, lateral to the supraorbital rim, just above the zygomatic-frontal suture line, the periosteum is elevated, and a hole is carefully drilled near the rim, through the ridge into the orbit (protecting the eye with a retractor). Wire is threaded through the opening and looped around the rim. The two wires are passed through the soft tissues downward under the temporal surfaces of the malar bone, the loose fragments are elevated into position, and the wires are fastened to the teeth. This holds the maxilla in place and also exerts an outward pull on the malar bone, holding it up in proper position. The wires can be left in position for several weeks, until bony union takes place. The wires attached to the maxilla are removed, and in order to prevent infection, the original incision of the skin is reopened at the orbital rim, the support wires are cut, and both strands are pulled downward and out through the mouth. This method is revolutionary, quick and simple in application, and no special apparatus is required. This new method will supplant the plaster head cap described by Federspiel [15] in a great many cases.

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12. Ivy, R. H., and Curtis, L.: Fractures of the Jaws, Philadelphia, W. B. Saunders Company, 1938.

13. Lothrop, H. A.: Fractures of the Superior Maxillary Bone Caused by Direct Blows Over the Malar Bone, Boston M. &. S. J. **154**:8, 1906.

14. Adams, W. M.: Internal Wiring Fixation of Facial Fractures, Surgery **12**:523 (Oct.) 1942.

15. Federspiel, M. N.: Maxillo-Facial Injuries, Wisconsin M. J. **33**:561 (Aug.) 1934.

COMPLICATIONS

Complications of a fracture of the malar-zygomatic compound will naturally involve adjacent structures such as the maxillary sinus, the nose, the orbit and the eyeball. Diplopia results from traction on the tendons of the oblique muscles. There may be injury of nerves, especially the infraorbital nerve. The antral wall is frequently crushed, and a hematoma in the maxillary sinus is common. Ivy and Curtis [12]

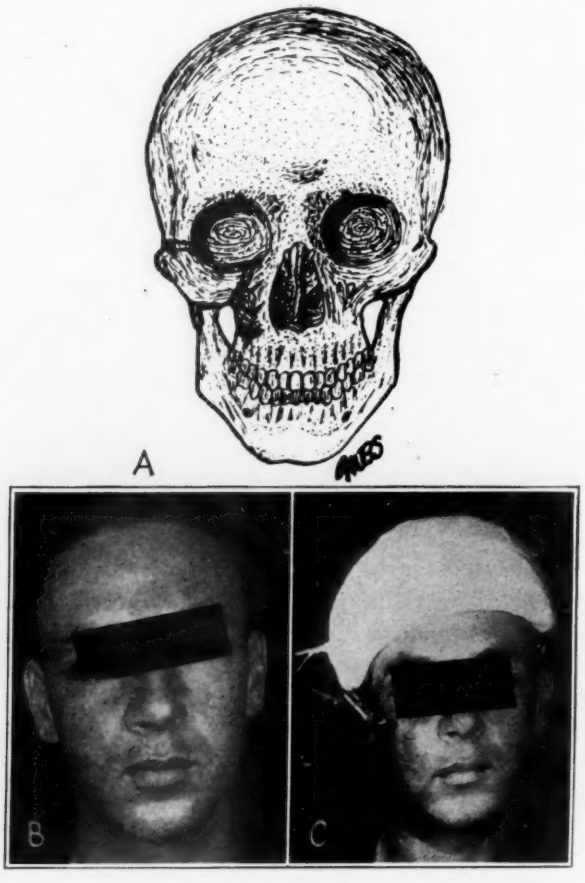

Fig. 3 (case 1).—A fracture of the malar bone with crushing of the antral wall. *A*, sketch of the fracture. *B*, the patient before surgical treatment. *C*, the patient after surgical treatment.

do not advise routine drainage of the antrum for this condition, except when suppuration takes place. In old fractures with deformity, a cosmetic defect can frequently be corrected by an implant of cartilage or by an open type of reduction. Incision across the facial fibers is sometimes necessary and the patient should be warned in advance

of such a complication. In old fractures of the malar-zygomatic compound contracture of the masseter muscle may prevent elevation. A special method for lengthening the masseter muscle which also saves the circulation is described in case 2.

REPORT OF CASES

CASE 1.—A private, a white man aged 22, was riding in an automobile when his car was struck by a drunken driver. The soldier was thrown against the car. The impact was sufficient to kill two other passengers. Several days later this patient was transferred to Tilton General Hospital. He had been unconscious for a short time and was admitted in good condition.

On inspection the patient had ecchymosis of the right eyelids with conjunctival hemorrhages. The right side of the face was swollen, but the infraorbital rim was depressed, and the fragment could easily be palpated. The malar bone was freely movable by palpation both outside and within the mouth. There was no external laceration, but there was contusion over the malar prominence.

The patient complained of pain but did not have diplopia. Roentgenograms showed fracture of the malar bone with some displacement downward and backward into the antrum. The outer wall of the antrum was comminuted.

Treatment.—By external manipulation the malar bone could easily be moved into position but would return to its original position of injury, downward and backward. External traction was planned. Inasmuch as the antral wall was badly crushed, the antral approach was used to reduce the malar bone, and the bony fragments were removed. The thick superior external wall of the antrum was used for anchorage, a hole was drilled, and a wire was looped through and brought out in two close strands through a small stab incision below the infraorbital crest. A plaster of paris head cap was constructed, and later traction was applied by rubber bands. A Caldwell-Luc operation was completed with an intranasal window, and the cavity was packed with iodoform gauze. The soft tissue flap was closed over the antrum, and the packing was removed through the nose in several days. After two weeks the traction wires and head cap were removed. Complete recovery resulted.

CASE 2.—A sergeant was admitted to Tilton General Hospital after having been treated in a lower echelon hospital for fracture of the skull and bones of the face and multiple fractures of the extremities. About a year previously the patient was riding on a motorcycle when he struck a 7 ton truck and was thrown for a distance of 40 feet (12 meters). The patient hovered between life and death for several weeks. There was a compound fracture of the zygomatic-malar compound with a wide and large open laceration over these bones. The patient's physical condition would not permit manipulation. The wound was filled with sulfanilamide powder and dressed frequently.

At Tilton General Hospital the patient exhibited a severe deformity of the left side of the face. The left malar bone was depressed about 1 inch (2.5 cm.) with a large external bowing of the zygomatic arch. Because of the wide scar directly over the displaced bones an external reduction was performed along with myoplasty.

With the patient under intratracheal anesthesia the large scar was excised from the side of the face. The operative area traversed many of the facial fibers. The misplaced bones were freed from their tissue attachments. Suitable holes were first drilled in the anterior and posterior parts of the future segment. The

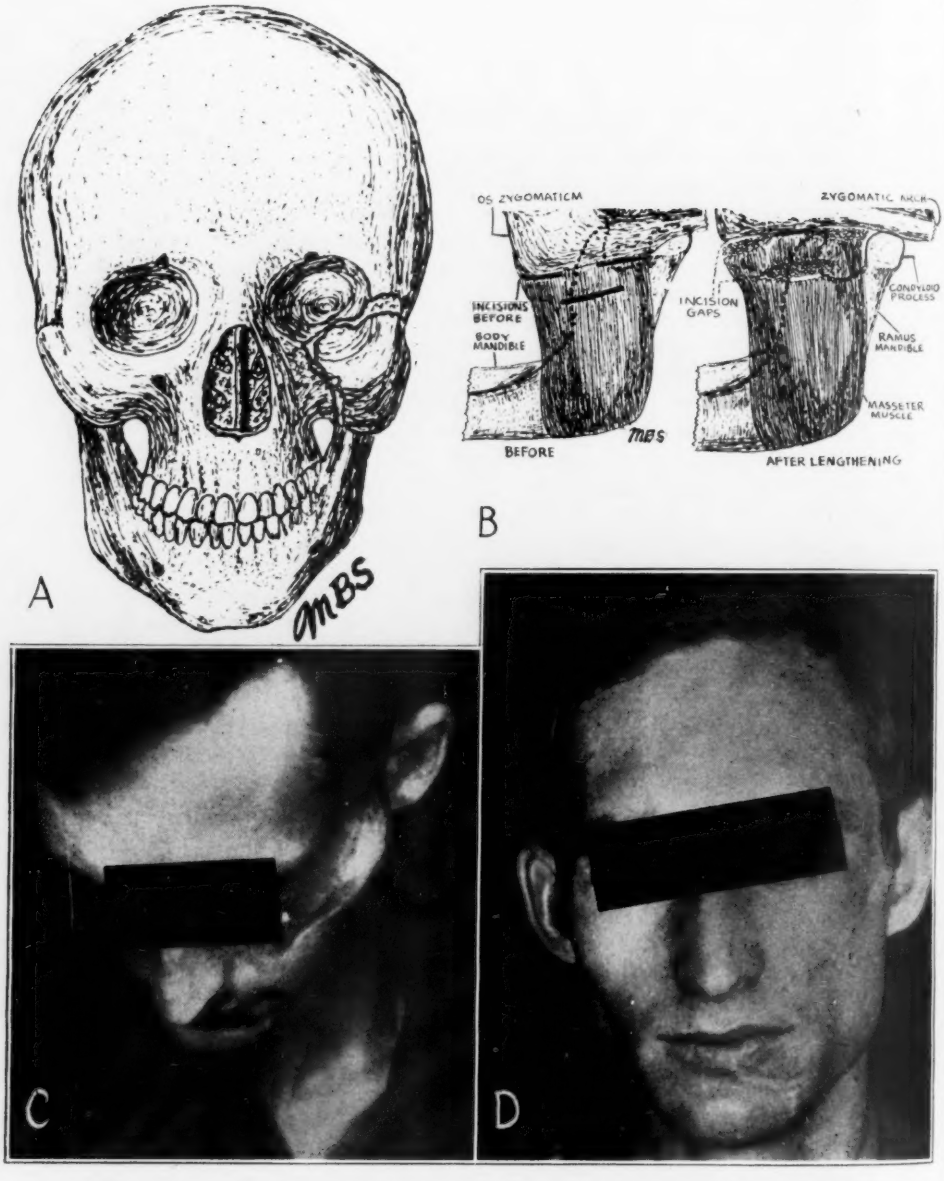

Fig. 4 (case 2).—Compound fracture of the malar-zygomatic compound. *A*, sketch of fracture. *B*, method of lengthening the masseter muscle. *C*, the patient before surgical treatment. *D*, the patient after surgical treatment.

misplaced bone was freed from its anterior and posterior attachments by the use of a saw. The contracted masseter muscle would not permit elevation of the now free segment; a myoplasty to lengthen the muscle was devised and performed as shown in the illustrations. The segment was wired to its new position with tantalum wire, and the wound was closed by deep and intradermal sutures. The cosmetic result was good, but weakness of the orbicularis muscle may necessitate future surgical treatment.

SUMMARY

This paper briefly reviews the history of fractures of the malar-zygomatic compound and emphasizes the most popular methods in use at the present time. Except when there are serious complications or infection, reduction of these fractures is in the hands of experienced operators and is simple and expedient. Post-traumatic deformities are usually caused by improper diagnosis and lack of treatment. Injuries to the face and jaws with interference in mastication and local tenderness indicate roentgen examination making use of the verticosubmental position. Usually no further information is necessary. Greater harm results when these fractures are allowed to unite in malposition; then more formidable measures must be employed. Almost routinely, for the past five years, we have used the method of Gillies and his colleagues for reduction of zygomatic fractures. With the collaboration of the Kings County Group 24 fractures of this type were corrected with good results, and no infection resulted when the method of temporal approach was used. Reduction of the malar bone may be done with good results by simple manipulation, by the methods of Lothrop, Ivy and Curtis. Sometimes an open reduction is done, with a cartilage or osseous implant when necessary.

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FREE VOICE AND PURE TONE AUDIOMETER FOR ROUTINE TESTING OF AUDITORY ACUITY

Studies on Comparative Efficiency

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NEW LONDON, CONN.

THE RELATION between a free voice acuity test and a pure tone audiogram is a complex one of many factors; no final statement has yet been made by which the interpretation of either can be made in terms of the other or by which both can be reduced to a common denominator. Some laboratories depend exclusively on a pure tone audiogram as a measure of how well a man can hear speech, while others look askance on the pure tone test and claim that only a test using speech sounds is valid as a measure of speech reception.

An early discussion of the relation between speech reception and audiometry is that of Fletcher,[1] who compared a free voice test and audiometry through the speech range (512 to 2048 cycles per second). Fletcher concluded, from the rather close correspondence of hearing loss, that the free voice and the pure tone audiogram measure essentially the same auditory function. It is clear that on the basis of laboratory data there is empiric justification for attempts to equate the two tests in routine situations.

However, when one comes to examine the relationship between the two measures as routinely obtained, one finds that in some cases little correspondence may exist. When, for example, in one experiment [2] the voice was placed at the low conversation intensity level, the authors concluded, "On the basis of our results, the spoken voice test may be summarily dismissed as of no diagnostic value."

But even when the voice is placed at the whispered intensity level, the screening out of persons with poor acuity through the speech range may be far from satisfactory, and, under conditions which generally prevail as to test chamber and training of personnel, the value of the whispered voice test may be expected to fall off sharply from maximum.

In any situation in which it is necessary to have the best available measure of acuity for speech, the worker, in deciding what test to use,

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From the Medical Research Laboratories, Submarine Base.

1. Fletcher, H.: Speech and Hearing, New York, D. Van Nostrand Company, Inc., 1929, chap. 6.

2. Shilling, C. W.; Everley, I. A., and Harris, J. D.: Hearing Tests: An Evaluation, U. S. Nav. M. Bull. **44**:100-116, 1945.

must consider such factors as the sort of sound-treated space available, the amount of testing time per man and the reliability and significance of the tests which it is possible to administer in the available space and time.

The present reference paper is designed to help the beginning worker survey his equipment and his needs and reach a decision as to which test or tests he should institute and to provide comparable data against which an experienced worker may check his own procedures.

The practical questions arise: (1) whether the free voice and pure tone audiometry actually are equivalent as measures of threshold acuity; (2) whether in a particular situation, with physical conditions less carefully controlled than in a laboratory, one test can be administered with more reliability and efficiency and so is to be preferred, and (3) if the two tests are of equal reliability, which test should be used to fit best the purposes of a particular activity. These questions will be considered in that order.

I. DO THE FREE VOICE AND PURE TONE AUDIOMETRY THROUGH THE SPEECH RANGE MEASURE THE SAME THING?

An extensively documented paper by Goldman[3] recently stated, "An analysis of the audiometric and whisper readings revealed that there was no constant correlation between the audiometric average decibel loss and the recording of the whisper perception. The same deficiency was noted between the audiometric and the low conversational voice tests." In view of such broad statements it is necessary at this time to examine the evidence on which Fletcher earlier concluded that the two tests were related and to examine later evidence bearing on the same point.

Fletcher's tables XXIV and XXV, pages 219 and 220, provide a comparison of hearing loss in decibels for 18 ears, first according to the free voice and again according to the average audiometric loss through the speech range. My colleagues and I have computed the correlation between hearing loss according to Fletcher's tests to be 0.95 ± 0.02. This is certainly a high degree of relationship. The standard error of estimate is 5.4 decibels, which means that by knowing the loss on either test one can predict loss on the other within 5.4 decibles about two thirds of the time. These figures provide strong grounds for the suppostion that the free voice and audiometry in the same range have not only a demonstrable but a high relationship when both are administered with laboratory care.

The trend of Fletcher's data for 18 ears is confirmed by a study from this laboratory on 335 ears, of which 126 scored below normal on the

3. Goldman, J. L.: A Comparative Study of Whisper Tests and Audiograms, Laryngoscope **54**:559-572, 1944.

whispered voice test. Thus the data need not be corrected for a restricted range of ability. The correlation between the whispered voice and the average audiogram loss from 512 to 2048 cycles per second was 0.69. All tests were conducted under less well controlled physical surroundings than Fletcher's data and under routine rather than experimental conditions, and the resultant drop in reliability could easily account for the drop of 0.26 in the correlation. Nevertheless the correlation of 0.69 is considered fairly high for such material, and it is probable that it is about as high a relationship as routine conditions will permit.

A more reliable voice test is one in which phonograph recordings replace the tester's own voice. In this case, hearing loss is measured directly in decibels rather than calculated from the voice distance fraction. One can find the relation between hearing loss according to the Western Electric 4-A phonograph audiometer and according to pure tone audiometry from Fletcher's same two tables. The standard error of estimate is 4.7 decibels. Evidently the relationship of the audiogram to the phonograph score is 0.7 decibels closer than to the whispered voice score. This increase in precision is almost certainly due to the increased reliability of the phonograph over the whispered voice.

Ciocco [4] likewise compared the average 4-A phonograph score with the average audiometer score at 1024 and 2048 cycles per second. For all groups whose loss averages between 0 and 50 decibels on the audiogram, a plot relating the 4-A score to the audiometer score can adequately be described as a straight line. An exact correspondence between absolute scores is not the case; rather, the phonograph score is approximately 0.6 that of the audiogram, but it is the linearity of the relationship which is important for the present argument.

In this laboratory, we have compared a series of 180 ears on pure tone audiometry and on a new recorded test of speech reception.[5] Among these 180 ears are found samples at all levels of acuity. A comparison of hearing loss according to these records and the average audiometer loss for 512 to 2048 cycles per second confirms Fletcher's experience in all details. On 94 ears with audiograms flat $\pm$ 5 decibels, we have found that the correlation between phonograph and audiometer scores is 0.93, with a standard error of estimate of 3.9 decibels. But even with 83 ears in which the audiogram varied more than 10 decibels (presumably those ears in which amplitude distortion would reduce intelligibility) we found a standard error of estimate of only 5.8 decibels.

4. Ciocco, A.: The Consistency and Significance of Tests Made with a 4 A Audiometer, Pub. Health Rep. **51**:1402-1406, 1936.

5. Manual of Instruction for Auditory Test No. 9: Threshold of Hearing for Words, Informal Communication number 73, NDRC Research on Sound Control, Psycho-Acoustic Laboratory, Harvard University, May 20, 1944.

Instead of the phonograph, a monitoring system for the voice may be used to control its intensity. The operator talks into a microphone, keeping his voice at constant intensity by watching a voltmeter and decreasing the output to the subject's loudspeaker by inserting resistance into the circuit. This technic was reported by Hughson and Thompson.[6] When their data are replotted in absolute terms, a precise linearity appears between speech reception and audiometry. Not only do their data plot in straight line fashion, but there is the closest correspondence between absolute scores—a loss of 50.3 decibels for speech is related to an average audiometer loss of 48.2 decibels and so on.

It has been thus seen that whether one considers the free voice (Fletcher and this laboratory), the phonograph voice (Fletcher and Ciocco, this laboratory) or the monitored voice (Hughson and Thompson) in general it is true that reception for speech and for pure tones in the speech range maintain a close parallel.

There is a certain error of prediction, which has been shown to be of the order of a few decibels, and there are one or two clinical types for which the correspondence may not hold but these considerations can hardly account for the cases in which speech and pure tone losses do not fairly well agree. In those cases one may reasonably look for some factor making for unreliability of one or both the tests. It is almost certain that in cases in which a worker finds a correlation lower than about 0.70 his speech reception scores or his audiometry data are less reliable than those possible even in routine military conditions such as ours. For 101 nerve-deaf ears reported by Goldman,[3] for example, the correlation between free voice and average audiometer loss at 256 to 2048 cycles per second was 0.24. But it can easily be shown (see figure 1) that in the test alley used by Goldman the tester had to move from 15 feet (4.5 meters) to within 2½ feet (0.7 meter) of the subject's ear before a loss of 5 decibels could be detected, while the maximum loss that could be detected even from the 6 inch (15 cm.) distance was about 15 decibels. His speech test, therefore, was not capable of wide enough range. We may agree with Goldman that in his test alley there was little correlation between speech reception and audiometry, but it is not necessary to apply this conclusion to other test situations.

Macfarlan [7] likewise stated, "There is no correlation between speech hearing and frequency hearing." However, when his data are plotted, it turns out that a correlation of 0.65 is present, while if his audiogram

6. Hughson, W., and Thompson, E.: Correlation of Hearing Acuity for Speech with Discrete Frequency Audiograms, Arch. Otolaryng. **36**:526-540 (Oct.) 1942.

7. Macfarlan, D.: Speech Hearing and Speech Interpretation Testing, Arch. Otolaryng. **31**:517-528 (March) 1940.

scores are multplied by 0.6 (the same factor found for Ciocco, it will be recalled) in only 6 of the 21 ears reported will the prediction of one score from the other be erroneous by more than 10 decibels. These are not striking figures, but at any rate it does seem from his own data that Macfarlan's conclusion of "no correlation" is premature.

The general conclusion to be drawn from the foregoing data is that for typical subjects and in cases in which vocabulary is controlled speech reception and pure tone audiometry are intimately connected and are concerned with measuring practically the same auditory function.

II. UNDER CONDITIONS OF TESTING WHICH ACTUALLY PREVAIL, WHAT ARE THE LIMITS OF RELIABILITY AND EFFICIENCY OF WHICH THE FREE VOICE AND AUDIOMETRY ARE CAPABLE ?

The conclusions of the first section of this paper are based largely on laboratory conditions. What must be discovered is how much reduction in reliability occurs when these tests are taken out of the laboratory and placed in the hands of routine testers under inferior and unsupervised test conditions.

A. The Reliability of Audiometry.—First the reliability of audiometry must be considered. Munson [8] compared thirty-eight test and retest scores on the Western Electric 6-A audiometer and reported the standard deviations of the differences between scores. He found that there were two chances in three that the retest would be within 4.3, 4.0 and 4.3 decibels for the respective frequencies 512, 1024 and 2048 cycles per second.

These figures, taken as they were, in a good laboratory under favorable conditions, may be taken as the precision of which a test using steps of 5 decibels intensity is capable. However, results as good as these have been obtained under service conditions. Giese,[9] on 109 subjects, reported standard deviations ranging from 3.6 to 4.6 decibels for the same frequencies as Munson. Harris,[10] for 64 subjects, reported data not differing essentially from these.

In terms of test-retest correlations, Giese's data show reliabilities from 0.63 to 0.73 and Harris' data from 0.70 to 0.87. In both of these groups these correlations are lower than would have been the case had a greater range of acuity been present. Even so, the data of Giese and of Harris compare favorably with the data of Witting and Hughson [11] on

8. Munson, W. A.: Trial Tests of Pulsing Tone Audiometer, unpublished research memorandum, Case 20871-2, New York, Bell Telephone Laboratories, Inc., 1937.

9. Giese, W. J.: Test-Retest Reliability of the Western Electric 6 B Audiometer Under Military Conditions, OSRD Report no. 91.11, Aug. 30, 1943.

10. Harris, J. D.: Group Audiometry, J. Acoust. Soc. America **17**:73-76, 1945.

11. Witting, E. G., and Hughson, W.: Inherent Accuracy of Repeated Clinical Audiograms, Laryngoscope **50**:259, 1940.

repeated audiograms. The latter's standard deviations of repeated audiograms on the same person (from 2.5 to 4.4 decibels) can be indirectly compared with data on only two audiograms but for more ears.

It may be concluded that pure tone audiometry as a general method, when performed by experienced operators under good room conditions with an audiometer in calibration and performing well, can easily satisfy minimum requirements as to reliability and as to uniformity of interpretation and that this may be accomplished in military situations.

When the retest is administered, in a second room, with a second tester using a different make of audiometer, the correspondence of test-retest will be somewhat less. We have made a comparison of audiograms taken routinely in our laboratory by group audiometry, using the Western Electric 6-B instrument, with audiograms on the same 192 ears taken routinely on a Maico audiometer about three months later at the West Coast Sound School, San Diego, Calif. The latter audiograms were furnished by Dr. Adelbert Ford. Although the mean acuity loss was about 10 decibels greater through the speech range for the data from San Diego (due in all probability to our more nearly sound-proof test chamber), nevertheless the correspondence between the two sets of data is satisfactory. In terms of the standard error of estimate, there were two chances in three of predicting the loss recorded at San Diego for any ear by 4.7, 4.3 and 5.3 decibels at 512, 1024 and 2048 cycles per second respectively.

The training and experience of the operator contribute somewhat to the reliability of an audiogram. For the most complete audiometry, involving bone conduction, localization and the like, only an operator with months of study under a good clinician will perform satisfactorily. But for routine audiometry, as, for example, in military screening, where most of the population is of normal or near-normal hearing, extensive training is not necessary. A study of test-retest reliability of three enlisted operators at this activity, where the retest was taken under the same conditions as the test (the operator being unaware that a retest was to be called for and a period of time elapsing to reduce incidental learning), shows that an operator can early attain a certain stability. In all cases these operators had had a week's informal lectures and demonstrations, had read most of Bunch's "Clinical Audiometry," [12] part 3 of Fletcher's "Speech and Hearing" [1] and chapters 1, 2, 10, 11 and 12 of Stevens and Davis' "Hearing." [13] The table shows how much average deviation exists between test and retest scores for a

12. Bunch, C. C.: Clinical Audiometry, St. Louis, C. V. Mosby Company, 1943.

13. Stevens, S. S., and Davis, H.: Hearing, New York, John Wiley & Sons, Inc., 1938.

sophisticated operator and for each of three operators given only a minimum of training and experience.

The procedure used by the experienced operator was one in which only two crossings of the limen were used in determining the final score. This procedure was taught to each operator as a minimum requirement; in case of doubt he was instructed to repeat readings. It is clear that there is a decrease in precision of 1 or 2 decibels for the enlisted operators but that with only slight training practically any intelligent person can learn to do audiometry on routine near-normal subjects with almost as good accuracy as a trained tester. It is my experience that more confidence can be placed in an audiogram taken by a technician with known personality traits of patience, accuracy and ability to put subjects at ease than in an audiogram taken by one whose chief qualification is that he has had a large amount of routine practice.

The general conclusion to be reached as to the limits of reliability of which audiometry is capable is that where space is provided with

Mean Deviation of Retest from Test Score, Western Electric 6-B Audiometer Being Used

Cycles per Second	Experienced Operator	Operator 1	Operator 2	Operator 3
512	2.30	3.40	4.50	3.40
1,024	2.00	4.15	4.05	2.35
2,048	2.10	4.85	4.25	2.80

reasonably low levels of noise only briefly trained technicans can furnish audiograms accurate to at least 5 decibels.

B. The Meaning and Reliability of the Free Voice Test.—In the case of the free voice test, the reliability statistics are not quite so encouraging. It is notorious that in cases in which a doctor is anxious that his therapy have some effect he is liable to the natural error of unconsciously raising his intensity level on a voice retest. But especially with routine testers the situation is critically bad. As often administered in the services, for example, the voice test screens out only the men with bilateral loss severe enough to require a hearing aid for normal conversation.

Of course, the fact that a test may be grossly misused does not mean that the test itself is valueless, but it must be emphasized that the free voice acuity test contains at best many sources of unreliability and that in cases in which conditions are not good its value even for rough screening is problematic.

It will serve no purpose to dwell on instances in which, owing to lack of supervision, the free voice test failed to detect a deafened ear.

What one needs to know is whether, given reasonable supervision, routine testers can perform satisfactorily. A measure of just how

satisfactory such testers can be is provided by data from this activity, in which a careful whispered voice test is given to all candidates for the submarine service. Every man sent to the submarine base has received a whispered voice score of 15/15 at the immediately preceding activity. This previously given test was administered under conditions as good as could be secured by frequent visits and communications from the officer in charge [14] of this activity. In these circumstances, the subsequent whispered voice test given here failed 68 of 1,555 supposedly qualified candidates, or 4.3 per cent, in a five month period. This figure represents a rather close correspondence between test and retest at different stations. From data obtained earlier, we know that had the whispered voice test not been given at the preceding station or been given in poorer fashion many more than 68 candidates would have had to be rejected for low auditory acuity.

For example, in one station from which the rejections at this activity were 5.7 per cent for a four month period, the whispered voice testing was found to be carried on in a reverberative metal Quonset hut; when this condition was corrected, our rejection of candidates from that station fell off to less than 1 per cent in the succeeding three month interval.

A check on the whispered voice test as conducted at this activity is provided by a careful 6 octave audiogram given routinely to all candidates for submarine service. For a typical three month period, out of 5,248 ears our whispered voice test labeled as normal 92 ears which had an average loss of 15 or more decibels through the speech range. Since an average loss of 15 decibels should correspond to a loss of several feet on an ideal voice test, these 92 ears, or 1.75 per cent, are an index of the error of selection of our voice test.

These data on the efficacy of the whispered voice show it to be of definite value as a quick screening device but show that even under the best service conditions an appreciable number of defective ears are overlooked.

In an effort to make the whispered voice score more meaningful, an experiment was undertaken to determine the "normal" distance for two types of phonetic material and for three types of test rooms. Five pharmacist's mates were used as testers, and 13 enlisted men with normal audiograms $\pm$ 5 decibels were used as subjects. Tests were conducted in a soundproof room 18 by 13 by 9 feet (5.4 by 3.9 by 2.7 meters) high, with attenuation from the outside in excess of 90 decibels; a classroom 29 by 21 by 10 feet (8.8 by 6.4 by 3 meters), with an average level of 50 decibels measured with a sound level meter, and, finally, out-of-doors, with sound level estimated at 35 to 40 decibels.

14. Captain C. W. Shilling (MC), U.S.N., Officer-in-Charge, Medical Research Laboratories.

The following tests were administered to each experimental ear by each tester in each room; in most instances a retest was run under so far as possible the same conditions:

1. Whispered voice, only the words "one," "two," "three," "four," "five," "six" and "eight" being used. Each tester was given directions consisting of those in the Manual of the Medical Department, U. S. N., 1939, plus the directions that at any distance the subject must repeat two out of three pairs of words correctly (the words to be said in pairs, thus: "one-three," "four-five," etc.) before the subject is given credit for that distance.

2. Whispered voice, a group of eighty-four spondees which have been shown to be of equal intelligibility being used.[5]

All the usual requirements of good experimental design and procedure were followed; testers were not told that all ears were normal in acuity or that their averages would be obtained; record sheets were collected from each tester as soon as he had finished with any subject on any test and similar requirements were fulfilled.

The results show that the designation of 15 feet (4.5 meters) as the denominator in the whispered voice fraction is approximately correct for typical Navy testing. For the digit list the test and retest mean distances in the classroom were 14.6 and 15.6 feet (4.4 and 4.7 meters) respectively, in the sound-proof room the distances were 16.1 and 15.8 feet (4.9 and 4.8 meters) and out-of-doors the mean test distance was 14.3 feet (4.3 meters). In these figures can be seen the tester's tendency to place his voice level in adjustment to the noise surround.

The same tendency can be observed in the case of the spondee list, for which in the sound-proof room the test and retest means were 14.9 and 14.8 feet (4.54 by 4.51 meters), while in the noisy classroom the retest mean was 14.2 feet (4.3 meters).

The effects of vocabulary and of learning in the whispered voice test are demonstrated by a difference in mean distance between test and retest scores for the spondee list in the classroom. On first exposure to these words the mean score was 11.3 feet (3.4 meters) (4.8 feet [1.5 meters] shorter than the first test using digits), but by the second test this distance was increased to 14.2 feet (4.3 meters), a figure agreeing well with the other data.

It may be concluded that a distance of 15 feet (4.5 meters) will, for a variety of test conditions and as an average of several testers, be found close to the "normal" distance.

To determine how close a tester can approximate a previous score on the same ear, one takes the average deviation of retest from test scores. The data from the classroom show that the chances are two in three that a tester can come within 3.7 feet (1.1 meters) for digit material and within 2.4 feet (0.7 meter) for the spondees when learning

is eliminated. The reliabilities for the sound-proof room are uniformly a little better.

A standard error of estimate of about 3 feet (0.9 meter) then expresses the precision of which this test is capable in routine hands. When expressed in decibels (see following material), it can be compared with the same datum for audiometry reported earlier in this paper.

Although every effort was made to simulate routine screening, the conditions of this experiment were nevertheless considerably better than ordinary ones, in the training of personnel, enthusiasm for testing and general supervision. Still, it is certainly fair to regard the precision reported here as within the reach of routine acuity testing.

The standard error of estimate of about 3 feet translates into only a few decibels (see figure 1) even for ideal room conditions. This compares favorably with the error reported here for audiometry and indicates that in cases in which supervision and test conditions approach ideal the acuity of normal or near-normal subjects can be placed with reasonable precision in routine screening.

C. Hearing Loss and the Acoustics of Sound Decay.—It is not possible any longer to defer a discussion of the error of predicting from the free field equation for hearing loss what will be the results from an actual testing chamber. According to the law that sound pressure falls off inversely as the square of the distance from the source, hearing loss $= 20 (\log d_o - \log d)$ where d_o is the distance at which a normal ear can just hear the sound used and d is the distance to be considered. But this equation is derived for a perfectly reflectionless room, which most acuity testing chambers assuredly are not. What is needed is an idea of the way in which sound actually does decay as a function of distance in typical testing chambers.

We have obtained an estimate of the decay of sound for three representative types of test rooms. The index used was the number of decibels a voice intensity had to be raised to maintain constant intelligibility as the sound moved away from the ear.

The specific procedure was as follows: A loudspeaker was set up 15 feet (4.5 meters) from a normal-hearing ear, and discrete words were presented by means of phonograph recording. An attenuation pad in 1 decibel steps was inserted between the phonograph output and the loudspeaker. The percentages of intelligibility for several attenuation readings were determined, and then the psychophysical function was drawn relating per cent intelligibility to attenuation reading; finally, from this curve, the attenuation reading corresponding to 75 per cent intelligibility was determined.

At this point the loudspeaker was moved to within 13 feet (3.9 meters), and again the attenuation reading corresponding to 75 per cent

intelligibility was determined. This process was repeated (suitably counterbalanced to control learning) for distances up to 6 inches (15.2 cm.).

The difference in attenuation readings for 15 feet and any other distance is found in decibels. It can be thought of as a measure of decay of ordinary speech through that particular distance and so can be compared with the theoretic computation of sound decay in a free field. Any discrepancy between a datum according to our method and the theoretic calculation for the same distance may be explained in terms of the lack of free field conditions in the particular test room used.

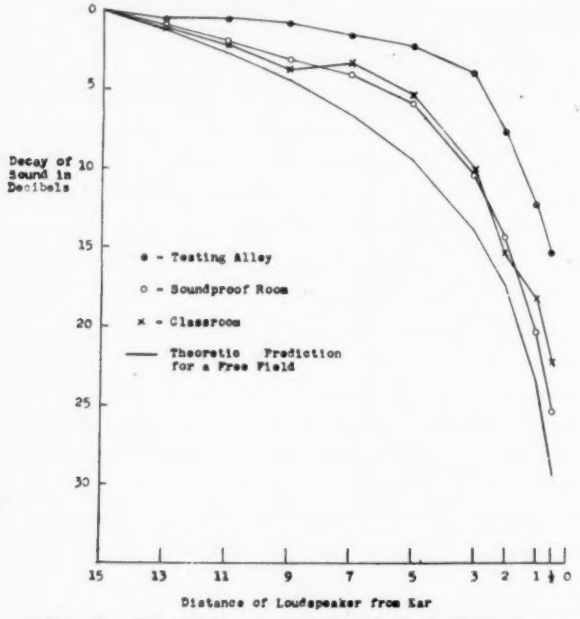

Fig. 1.—Sound decay as a function of distance.

It is of interest to note that the datum described here can also be taken as the decibel hearing loss corresponding to a whispered voice distance fraction. As the sound source moves from 1 to 15 feet (0.3 to 4.5 meters) from the ear, it must be made so many decibels louder to maintain constant intelligibility. But it is equally correct to say that an ear deafened by that same amount will have to be within 1 foot of a sound which a normal ear could hear at 15 feet. We have in this technic and in this chain of reasoning a rational empiric basis for building a conversion table for voice and for audiometry data. I hope to develop this possibility in a succeeding paper.

The necessity of taking actual measurements of sound decay in a test room rather than relying on a theoretic prediction is illustrated in figure 1. Here are shown the data for three test rooms. At the 15

foot distance, the sound decay is, of course, zero; then, for any shorter distance the decay is read on the ordinate directly in decibels.

The solid curve in figure 1 is the theoretic prediction for free field; the open circles for our sound-proof room; the closed circles for our voice test alley, a wooden, painted room 26 by 5 by 9¼ feet (7.9 by 1.5 by 2.8 meters) high, and the crosses for a classroom 30 by 21 by 10 feet (9.1 by 6.4 by 3 meters) high. Present in the sound-proof room the classroom and the alley was a white noise registering respectively 50, 52 and 58 decibels on a sound level meter at the position of the ear. This ambient noise was introduced merely to provide a uniform mask. It was not loud enough to induce serious harmonic distortion in the acoustic analyzer and should have no effect on the shape of curves in figure 1.

The difference between sound decay in the free field and in the sound-proof room is never greater than 4 decibels at any distance. What is even more surprising is the close correspondence between the sound-proof room and the acoustically untreated classroom, the latter being only 3 decibels worse at most and then only for the very near distances. Throughout the greater part of the distance the two rooms yield indistinguishable results.

The data for the narrow test alley diverge much more from the free field equation. Relatively little sound decay occurs through 14 feet (4.2 meters)—only about 12 decibels.

The practical significance of these facts can easily be shown: if, for example, the ambient noise in a test chamber raises the threshold by 20 decibels, the ears suffering loss up to 20 decibels cannot be detected from normal; now with a voice intensity constant at $d_o = 15$ feet, figure 1 shows that even in a free field a range of only 29.5 decibels would be available, and this is reduced to only about 24 decibels in an actual room. It is true, therefore, that in a room causing a threshold shift of 20 decibels with a whispered voice constant at $d_o = 15$ feet only the ears with losses of 20 to 44 decibels could be placed at all reliably on an acuity scale. Persons with losses less than 20 decibels would be reported perfectly normal, though some would have difficulty hearing in other situations, and those with losses greater than 44 decibels would be uniformly classed as completely deaf, though many of them would have usable residual hearing.

We know, however, from a vast amount of clinical data from this and other laboratories, that testers can and do consistently screen out ears which show loss of less than 20 decibels and can distribute on an acuity continuum the ears which have worse than a 44 decibel loss.

It is obvious that to do these things some variable other than distance is used. The tester must have changed the intensity of his voice in some systematic fashion.

In such cases, in which a tester uses both distance and voice intensity as variables, the foregoing calculations of test reliability do not apply. It must be emphasized that the present conclusions as to the reliability and usability of a free voice test apply only to a normal or near-normal population and not to a population of seriously deafened ears. For such testing, the error of prediction will be greater; in order to determine the amount of increase of error, further calculations would be necessary.

D. Voice Intensities Necessary for an Effective Free Voice Test.—From an article by E. P. Fowler Sr.,[15] it is possible to calculate the voice intensities required in order to use the voice as an instrument for testing seriously deafened ears. Free voice distance scores are given for 17 ears, together with complete audiograms. From the distance at which the voice could just be heard and from the audiometric hearing loss, the voice intensity used can be calculated in terms of how far a normal ear could hear that voice. The assumptions on which this calculation is based are all well documented in the present paper.

Figure 2 shows the variation in voice intensity used for the seriously deaf. Although only 1 to 4 ears are included at each distance, a fairly regular tendency is seen for the test voice to become louder as the ears tested are poorer in acuity. The tendency may be summarized by stating that the log of the voice intensity varies inversely with the voice score. In absolute terms, the intensity used to produce a 20 foot (6 meters) voice score could be heard by a normal ear at 43 feet (13 meters) (log 1.6335), and at the other extreme the intensity used to produce a 1 foot score could be heard at 2015 feet (614 meters) (log 3.3043).

It is evident that the accurate placement of the voice at intensities from a low whisper to a loud shout is beyond the ability of any but the most experienced tester and that one could not reasonably expect any reliability on such a test with routine testing personnel.

E. Relative Testing Times.—Another measure of the relative efficiency of the two tests is the amount of testing time per man. Obviously in clinical work the element of time is of secondary importance, but there are activities processing thousands of men in which a saving of even half a minute is of significance.

The longest testing time is taken by individual audiometry. A procedure in which each threshold is crossed at least twice before the final reading is taken being used, an average of 4.5 minutes is needed to perform a 6 octave audiogram on the 2 ears of a subject of average or better intelligence and near-normal acuity. This figure will predict

15. Fowler, E. P., Sr.: Hearing Standards for Acceptance, Disability Rating and Discharge in the Military Services and in Industry, Laryngoscope **51**:937-956, 1941.

rather well how many such audiograms can be completed in a specific period.

For the same type of subject, the whispered voice individual examination as we routinely administer it takes thirty-four seconds for the 2 ears. This mean time includes a range of ten to one hundred seconds for several dozen subjects.

Two modifications of audiometry are available to effect considerable saving of time, the sweep frequency technic and group testing. Either of these will prove more economical of time than the free voice examination under certain conditions. When all that is wanted is assurance that an ear can hear within 10 decibels of normal at all frequencies, it is sufficient to set the attenuation dial at 10 decibels and then to

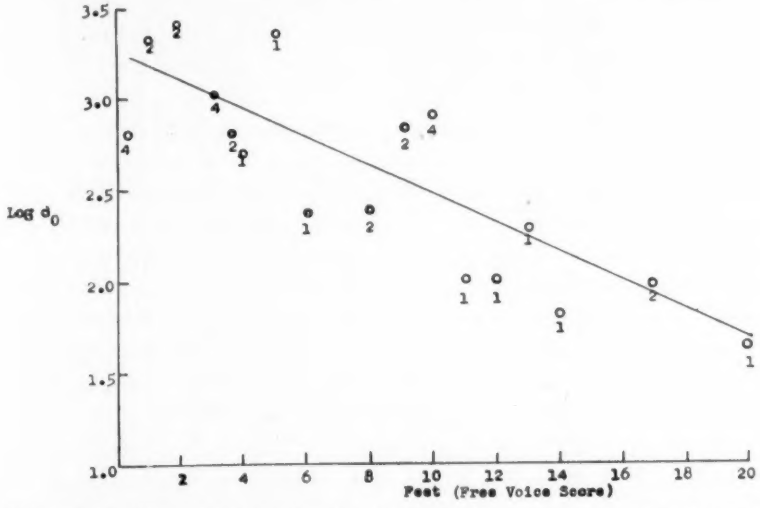

Fig. 2.—Relation of voice intensity to free voice score (calculated from data of E. P. Fowler Sr.). Note that numerals indicate the number of cases. Log d_0 indicates the distance at which the normal ear could hear a voice which produced a certain free voice score. For example, a free voice intensity which resulted in a score of 10 feet (average audiometer loss of 4 subjects of 38.2 decibels) could be heard a distance of 813 feet (log 2.9101) by a normal ear. An intensity resulting in a score of 20 feet (average loss of 6.6 decibels) could be heard a distance of 43 feet (log 1.6335) by a normal ear.

sweep through the desired frequency range, interrupting the tone at appropriate points. By this means a satisfactory 6 octave screening audiogram can be performed on both ears in an average time of one minute and thirty-eight seconds.

If phones comparable in output are obtainable, a group of them can be coupled to the output of almost any commercial audiometer and complete audiometry performed on a number of subjects simultaneously.[10] We have found that a 6 octave audiogram for both ears can be performed on 12 subjects in twenty minutes on the average, or about one

minute and forty seconds per man, but there is no reason except lack of space or equipment why any number of phones should not be used and testing time cut to less than a minute per man.

In comparison of these audiometer testing times with those for the free voice, it must be remembered that the two types of test are not quite comparable, since all audiometer times given here are for 6 octaves, while the voice test provides pertinent information only for 3 octaves. If audiometer times were on a 3 octave basis, a time saving of perhaps a third would be effected.

III. GRANTED THAT AT A PARTICULAR ACTIVITY EITHER TEST COULD BE SET UP SATISFACTORILY, SHOULD ONE OR BOTH BE USED?

Since a free voice test and a pure tone test may be considered as interchangeable for routine screening when both are given under conditions approaching ideal, it remains to consider which should be recommended for a particular activity.

It is obvious that for a complete survey, as in clinical work, or for the fitting of hearing aids both speech and pure tones must be used, or else in some cases important information will be overlooked. It is also perfectly obvious that for certain job requirements only an extended frequency range, pure tone audiogram will suffice. But for the vast majority of acuity testing—screening for the armed services, Veteran's Administration, civil aviation or industry, where all that is required is a fair ability to hear human speech—only one type of test is necessary. The decision for any such activity need be based only on which test can be set up most efficiently and reliably.

Enough data have been presented in this paper to provide a basis for decision in some cases. For example, if group audiometry is possible for 36 subjects simultaneously, it will result in saving of time even over the most cursory free voice test and provide, in addition, valuable information on hearing at frequencies above and below the speech range. If audiometry must be individual, the free voice is quicker and under good conditions can, by careful supervision, be made almost as satisfactory as audiometry for the screening of a near-normal population. However, if the usual audiometric procedure is modified to a screening technic, whereby the operator merely assures himself that the hearing of a subject is better than a certain critical level, then individual audiometry is almost as rapid as a free voice test if even a little care is taken with the latter. Again, if testing must be accomplished on a population suffering generally from defective hearing, then the present paper makes it clear that there are factors at work which sharply affect the reliability of a free voice test and, unless rigorously controlled, are capable of reducing its meaning nearly to zero. On the other hand, while the precision of audiometry is reduced somewhat

with a deafened population, this reduction is of the order of a few decibels at most and is not to be compared with the error of prediction of a free voice test carried out close to a subject's ear. With such a population, audiometry in preference to the free voice should be mandatory. Other applications of the present data can be made to fit other needs.

SUMMARY

A reference paper on auditory acuity testing is presented, designed to help the beginning worker reach a decision as to which test or tests he should institute and to provide a body of data against which the experienced worker may check his own procedures.

Data are summarized from this and other laboratories bearing on the relative usability of the free voice and the pure tone audiometer as tests of auditory acuity. It was found the consensus of most studies that a careful voice test and pure tone audiometry through the speech range (512 to 2048 cycles per second) are intimately connected and measure almost the same auditory function. A comparison of six studies reveals the relationship between the two tests to be a straight line function. Statements cited to the effect that speech and audiometry are not related are definitely contraverted.

Recent studies on the reliability of these tests show that routine audiometry can well be expected to be accurate to within 5 decibels and that for near-normal subjects the reliability of the whispered voice need not be less.

A distance of 15 feet (4.5 meters) can be depended on to be a fairly accurate "normal" distance for the whispered voice, regardless of room acoustics and noise level.

A novel method of measuring sound decay as a function of distance is presented. By means of this method, the acoustic characteristics important for a voice test were compared with free field conditions for three representative test rooms. It was found that the usual narrow, reflective alley is almost useless for acuity testing. It was determined that for a population of near-normal acuity a whispered voice test in a fairly nonreflective room could serve but that for a seriously deafened population the additional variable of voice intensity would have to be added to that of distance.

Data from Dr. E. P. Fowler Sr. are analyzed to show that for a deafened population it is necessary to vary the voice from an average whisper to a loud shout. The log of voice intensity varies inversely with distance score. It is concluded that such control is beyond what can be expected from routine testers.

A summary is given of relative testing times for several modifications of the acuity test.

A statement is given of the relative applicability of voice and pure tone testing for a variety of specific situations.

LOCAL USE OF PENICILLIN AND OF SULFATHIAZOLE FOR ACUTE AND CHRONIC SINUSITIS

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THE WIDE use of penicillin and the favorable results of its intramuscular injection and in certain cases of its topical application indicated that its local use for sinusitis might prove beneficial.

Since December 1944 my colleagues and I have been investigating the effects of penicillin instilled into the maxillary sinus after irrigations in cases both of acute and of chronic infections. Starting with a dilution of 250 units per cubic centimeter, we increased the concentration of penicillin to 500, 1,000, 2,000, 2,500, 5,000 and 10,000 units per cubic centimeter in isotonic solution of sodium chloride without impressive results. At the present time, after irrigation of the antrum and displacement of the isotonic solution of sodium chloride with air, 2 cc. of isotonic solution of sodium chloride containing 10,000 units of penicillin per cubic centimeter is instilled and allowed to remain in the sinus. No evidences of irritation or of sensitivity have been encountered so far. However, in the series of cases reported, penicillin has effected a cure in only 1 case in which a solution of sulfathiazole had previously failed. The patient, a man aged 45, giving a history of five years' duration, had a severe chronic infection of the right maxillary and ethmoid sinuses. The organism was Staphylococcus aureus. The antrum was filled with a foul necrotic purulent discharge and a Caldwell-Luc operation was advised. The patient refused this and also an antrotomy. Subsequent irrigations with instillations of sulfathiazole, tyrothricin and Pickrell's solution (a solution of sulfadiazine in triethanolamine) produced only slight improvement, but seven instillations of 5,000 units of penicillin each effected a cure.

Soon after Turnbull reported his use of sodium sulfathiazole for sinusitis,[1] we investigated treatment with a freshly prepared 5 per cent solution of sodium sulfathiazole instilled into the maxillary sinuses after irrigation. This was applied in cases both of acute and of chronic sinusitis with satisfactory results; however, this highly alkaline solution produced local reactions and caused the patient discomfort. When, sometime later, a stable solution of 2.5 per cent sodium sulfathiazole

1. Turnbull, F. M.: Intranasal Therapy with Sodium Sulfathiazole in Chronic Sinusitis, J. A. M. A. **116**:1899-1900 (April 26) 1941.

in combination with a vasoconstrictor, 0.125 per cent *dl*-desoxyephedrine hydrochloride, became available, the latter solution was substituted. It had the advantages of stability and of not requiring preparation for each patient. There was an additional distinct advantage in its vasoconstrictor content, and it seemed to produce much less irritation in patients, both subjectively and in relation to reaction of the nasal mucosa.

Three years' experience with the use of this compound has shown that it produces highly satisfactory results in the treatment of acute and chronic sinusitis, and in acute coryza in the presence of secondary invaders or of a purulent discharge. Comparative studies with penicillin have created the impression that this substance is not so satisfactory for such infections. Frequently two or three treatments with sodium sulfathiazole will effect a cure, whereas a greater number of treatments with penicillin will not. In other instances, patients have received treatment with penicillin without improvement but have recovered after being transferred to the other compound.

As is true of infections elsewhere in the body, it seems rational to select the compound most efficacious for the specific organism or strain being dealt with. It would seem logical that sulfadiazine might succeed where sulfathiazole failed. However, the results with Pickrell's solution, which contains sulfadiazine but no vasoconstrictor, were not so impressive in these cases as the results with sulfathiazole containing the vasoconstrictor. This is not surprising if one takes into consideration the intense edema of the mucous membrane lining the sinuses in an acute infection. The introduction of a solution containing a vasoconstricting agent produces decongestion and shrinkage of the mucous membrane, thus exposing a greater surface area to the bacteriostatic sulfonamide component.

Tyrothricin has also been investigated, as a 1 per cent solution in isotonic solution of sodium chloride. In approximately 50 instances of antrum irrigations and of sinus irrigations in acute coryza, the results were not sufficiently impressive to warrant the continued use of this substance.

MODE OF TREATMENT

The sinus is irrigated with isotonic solution of sodium chloride; the solution is displaced by air, and the sinus cavity is then completely filled with a 2.5 per cent solution of sodium sulfathiazole in combination with 0.125 per cent *dl*-desoxyephedrine hydrochloride. In most cases the instillations are made through the natural opening with the Bowers cannula.

In cases of acute sinusitis the irrigations and instillations of sulfathiazole are given daily or at frequent intervals. When penicillin is

used, instillations are made daily in an effort to keep the therapeutic agent in contact with the infecting bacteria and to lessen the likelihood that the organism will become resistant to penicillin.

Twenty-four hours after irrigation the reddish pigment in the sulfathiazole compound and the yellowish pigment of penicillin can be recovered. This indicates that both compounds are retained in the antrum for a sufficient time to exert a bacteriostatic effect.

EXPERIENCE WITH SOLUTION OF SODIUM SULFATHIAZOLE

Several published reports have indicated that sodium sulfathiazole is effective in the treatment of acute and chronic sinusitis and for relieving the congestion of the nasal passages generally associated with the common cold.[2] Dosage is 4 to 5 cc. by instillation following irrigation and displacement by air. In the common cold, when neither secondary infection nor pus is seen, it is questionable whether this treatment has influenced the course of the infection. However, in the presence of secondary invaders, or when a purulent discharge was present in the upper part of the nose or in the middle meatus, the local application of the preparation was most beneficial. Patients with acute or chronic ethmoiditis showed good response to treatment when the sodium sulfathiazole compound was instilled into the antrum. The prolonged and gradual escape of this bacteriostatic and vasoconstricting agent into the middle meatus has given increased drainage of the ethmoid cells and has exposed them to more prolonged contact with a bacteria-inhibiting compound. The desoxyephedrine component is valuable because it causes decongestion and thus permits a greater amount of the therapeutic agent to penetrate to the affected membranes and to exert its local bacteriostatic effect.

Two reports of cases are submitted to illustrate the greater effectiveness of sulfathiazole as compared with penicillin.

REPORTS OF CASES

CASE 1.—The patient (D. P.) sustained a crushing injury of the frontal and maxillary sinuses in an automobile accident ten years ago. These sinuses became infected, and Caldwell-Luc and Lynch operations were performed. The frontal sinus failed to heal, and a draining fistula above the left superciliary arch leading into the left frontal sinus persisted. The patient refused further surgical treatment and requested instillations of penicillin. The sinus was irrigated with isotonic solution of sodium chloride through the fistulous tract, by means of a blunt lacrimal duct needle. About 5 cc. of thick creamy pus was returned.

2. Fenton, R. A.: Local Use of Sulfathiazole in Otolaryngologic Practice, Arch. Otolaryng. **37**:491-495 (April) 1943. Freund, E. M.: Local Use of Some of the Newer Sulfonamide Solutions in Diseases of the Ear, Nose and Throat, Eye, Ear, Nose & Throat Monthly **23**:104-106 (March) 1944.

A culture of this showed the presence of Micrococcus catarrhalis and a gram-negative bacillus. A solution of sodium iodide was instilled into the sinus, and roentgenograms were taken. They showed no evidence of osteomyelitis and no communication between the frontal sinus and the nose.

Eleven subsequent irrigations with isotonic solution of sodium chloride, displacement of the solution and daily instillation of 5,000 units of penicillin produced no improvement, and the cultures remained positive. Irrigations with aqueous solution of merthiolate and with aqueous solution of phemerol were also ineffective. Three instillations of sodium sulfathiazole produced a complete cessation of discharge. One month after the last instillation of sulfathiazole the cavity was entirely free from discharge. Thus three instillations of sulfathiazole apparently accomplished what eleven of penicillin failed to do.

CASE 2.—The patient (W. M.) had had five recurrent acute attacks of sinusitis in the left maxillary antrum, starting in January 1943. Five irrigations were done through the natural opening with isotonic solution of sodium chloride, producing moderate improvement. This was followed by instillation of 6 cc. of a 5 per cent solution of sodium sulfathiazole, which produced clear returns on irrigation of the antrum in three days. One week later the sinuses were perfectly clear on transillumination. They remained so until another acute attack in September, when the sinus was irrigated with isotonic solution of sodium chloride and filled with a 2.5 per cent solution of sodium sulfathiazole combined with 0.125 per cent *dl*-desoxyephedrine hydrochloride. This treatment was repeated two days later. A third irrigation four days later showed clear returns. One week later the sinuses were perfectly clear to transillumination and remained so until April 1944, when the patient contracted acute coryza. Both antrums were irrigated twice, at a two day interval, and returned considerable mucoid pus. The solution of sodium sulfathiazole and *dl*-desoxyephedrine was instilled three days later. Ten days after this the patient was seen again, and the sinuses were perfectly clear. In October there was an acute flare-up of the left antrum following another attack of coryza. The same solution was instilled October 27 and 30, and on November 3 irrigation showed clear returns. Again the patient remained well until he contracted an acute infection of the upper respiratory tract, in May 1945. The left antrum was cloudy to transillumination, and 2 drachms (7.8 Gm.) of pus was recovered on irrigation. Penicillin, 20,000 units, was instilled on May 18, 20, 22, 25 and 29, with only gradual improvement until June 1, when returns from washing the antrums were clear. Thus, in this patient five irrigations of the antrums with penicillin produced the same results as one or two with the solution of sulfathiazole. The organism in this case was a pneumococcus.

Such experiences seem to be in agreement with Kolmer's statement [3]: "It will be observed that with the exception of staphylococcal septicemia, penicillin has not proven superior or but slightly superior to the sulfonamide compounds commonly employed at the present time."

Sale and Diamond,[4] advocating the trial of penicillin for sinusitis, reported that they had cured chronic bilateral maxillary sinusitis of seven

3. Kolmer, J. A.: Penicillin Therapy, New York, D. Appleton-Century Company, Inc., 1945, p. 163.

4. Sale, G. G., and Diamond, E. H.: Chronic Suppurative Maxillary Sinusitis Treated Locally with Penicillin by a New Method, Arch. Otolaryng. **40**:406-408 (Nov.) 1944.

or eight years' duration by implanting a spinal puncture needle into each antrum and instilling 5 cc. of a solution containing 1,000 units of penicillin per cubic centimeter every three hours for eight doses.

TABLE 1.— *Results of Treatment with Sodium Sulfathiazole and Dl-Desoxyephedrine Hydrochloride*

Patients with:	Cured	Not Cured
Acute maxillary sinusitis	35	3
Subacute maxillary sinusitis	8	0
Chronic maxillary sinusitis	13	6
Chronic frontal sinusitis	1	0
	56	9

TABLE 2.—*Results of Treatment with Penicillin*

Patients with:	Cured	Not Cured
Acute maxillary sinusitis	9	7
Subacute maxillary sinusitis	2	0
Chronic maxillary sinusitis	3	8
Chronic frontal sinusitis	0	1
	14	16

TABLE 3.—*Type of Bacterial Infection Encountered*

	Acute	Subacute	Chronic
Pneumococcus	21	2	4
Staphylococcus albus	13	3	6
Staphylococcus aureus	9	0	8
Streptococcus haemolyticus	3	1	0
Streptococcus nonhaemolyticus	5	4	5
Streptococcus viridans	2	1	2
Micrococcus catarrhalis	3	1	1
Gram-positive cocci	0	0	1
No growth	2	1	0
Short gram-positive rod	0	0	1

TABLE 4.—*Average Number of Instillations Needed to Effect a Cure*

	Acute Infection	Chronic Infection
Sulfathiazole	3.4	6
Penicillin	4.2	6

This twenty-four hour treatment was repeated on the twelfth day. It is questionable whether this treatment is not of more inconvenience to the patient than antrotomy.

Results with sulfathiazole and with penicillin are given in tables 1 and 2. The maxillary sinus was flooded with sulfathiazole at each treatment when this compound was used. In a sizable series of cases there was a good response to instillations of sulfathiazole, but the only cases included in the tabulation are those in which cultures were taken.

SENSITIVITY

It had often been suggested that the use of the sulfonamide compounds should be reserved for severe illnesses to avoid the danger of producing sensitization. Coburn,[5] in summarizing the prophylactic use of sulfadiazine among 600,000 Naval personnel, in which 1 Gm. was given daily for as long as three to six months, concluded that sensitization did not appear to have been induced by previous use of the compound. He found that among men receiving 1 Gm. of sulfadiazine daily the incidence of mild reaction was approximately 0.5 per cent and that only one half of these reactors were sensitive on being retested with 1.0 Gm. of sulfadiazine. Coburn further stated, "The subjects who manifested reactivity to sulfonamide prophylaxis after the first day gave no history of previous sulfonamide therapy."

In our entire series of cases, no outstanding allergic or irritative reactions following instillations of either sulfathiazole or penicillin have been noted.

SUMMARY

1. This series of 95 cases of acute and chronic maxillary sinusitis indicates that a solution of sodium sulfathiazole exerts a bactericidal effect when instilled into the sinus cavities.

2. A vasoconstricting agent in combination with a sulfonamide compound is desirable in the treatment of the infected mucous membranes.

3. Penicillin instilled into the antrums appeared to be less effective than sodium sulfathiazole containing a vasoconstricting agent.

11 Wall Street.

5. Coburn, A. F.: Mass Sulfadiazine Prophylaxis of Respiratory Disease in the U. S. Navy, Bull. New York Acad. Med. **21**:281-301 (June) 1945.

EFFECT OF SULFONAMIDE DRUGS ON MUCOUS MEMBRANES

An Experimental Study of the Action of the Newer Sulfonamide Drugs on the Mucous Membranes of Rabbits

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IN APRIL 1941 Turnbull [1] described the favorable use of sodium sulfathiazole sesquihydrate in 45 out of 47 cases of infection of the nose and throat. After his article had been published, a number of other writers, using either powdered sulfanilamide and sulfathiazole or sodium sulfathiazole in solution, reported good results (Marks,[2] Ebert,[3] Connell and Trowbridge,[4] Silcox and Schenck,[5] Brown,[6] Merica,[7] Fenton [8] and Ballenger [9]).

During this time Fletcher [10] described a case in which repeated washing with sodium sulfathiazole destroyed the mucosa of the antrum.

From the Department of Otolaryngology, Baylor University College of Medicine.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1945.

1. Turnbull, F. M.: Intranasal Therapy with Sodium Salt of Sulfathiazole in Chronic Sinusitis, J. A. M. A. **116**:1899 (April 26) 1941.

2. Marks, R. F.: Sulfathiazole in the Treatment of Sinusitis, Arch. Otolaryng. **35**:794 (May) 1942.

3. Ebert, E.: Local Treatment of Acute Rhinitis with Sulfathiazole, Arch. Otolaryng. **38**:324 (Oct.) 1943.

4. Connell, E. S., and Trowbridge, B. C.: Local Use of Sulfanilamide and Its Derivatives in the Ear, Nose and Throat, Arch. Otolaryng. **35**:740 (May) 1942.

5. Silcox, L. E., and Schenck, H. P.: Use in Otolaryngology of Microcrystals of Drugs of the Sulfanilamide Group, Arch. Otolaryng. **36**:171 (Aug.) 1942.

6. Brown, L. A.: Intrasinal Application of Sulfonamide Powder, Ann. Otol., Rhin. & Laryng. **51**:611, 1942.

7. Merica, F. W.: The Use of Sulfonamide Solutions in the Nose, Correspondence, J. A. M. A. **119**:286 (May 16) 1942.

8. Fenton, R. A.: The Local Use of Sulfathiazole in Otolaryngologic Practice, Arch. Otolaryng. **37**:491 (April) 1943.

9. Ballenger, H. C.: The Local Use of Powdered Sulfathiazole in Acute Naso-Pharyngitis, Quart. Bull., Northwestern Univ. M. School **18**:109, 1944.

10. Fletcher, R.: The Caustic Action of Sodium Sulfathiazole, Correspondence, J. A. M. A. **117**:1204 (Oct. 4) 1941.

In experimental work on this problem, Futch, Rosenvold and Stewart [11] found that 5 per cent sodium sulfathiazole destroyed the mucosa of rabbits in which it was used for two to ten days. Gundrum,[12] working with 4.7 per cent sodium sulfathiazole, 4.7 per cent sodium sulfadiazine and 4.7 per cent butanoylsulfanilamide, reported that all three drugs produced a sloughing of the mucosa of rabbits in which it was used ten or thirty days. Hunnicutt [13] tried 1, 3 and 5 per cent sodium sulfathiazole on Swiss mice. He also found that the mucosa was damaged, though the mice had a tendency to recover if the administration of drops were continued over ten days.

Most of the authors, in considering the ill effects of the various sodium salts of the sulfonamide drugs, stated the belief that the damage was done by the excessive p_H of the solutions, which is around 10. Proetz,[14] in his book, stated that the normal p_H of the nose is from 7.6 to 8.4. Negus [15] found that cilia were most active when the solution had a p_H of 8.5. However, Fabricant [16] stated that the p_H of the nose normally is from 5.5 to 6.5. Van Alyea [17] approved of shrinking solutions with a p_H around 5.5 to 6.5.

After the reports given came out, the various pharmaceutical houses developed a sulfonamide solution with a lower p_H, or made a suspension of microcrystals of sulfathiazole. These have been on the market some time. Several authors have written in favor of their use (Freund [18]; Crowe and Ward [19]; Silcox and Schenck [5]; Turnbull, Hamilton, Simon

11. Futch, C. E.; Rosenvold, L. K., and Stewart, C. E., Jr.: Caustic Effect of Sodium Sulfathiazole Solution on Nasal Mucous Membrane, J. A. M. A. **119**: 7 (May 2) 1942.

12. Gundrum, L. K.: Effect of the Newer Sulfanilamide Derivatives on the Nasal Mucosa of Rabbits, Arch. Otolaryng. **37**:209 (Feb.) 1943.

13. Hunnicutt, L. G.: Reaction of Mucous Membranes to 5 Per Cent Solution of Sodium Sulfathiazole, Arch. Otolaryng. **36**:837 (Dec.) 1942.

14. Proetz, A. W.: Applied Physiology of the Nose, St. Louis, Annals Publishing Company, 1941, p. 269.

15. Negus, V. E.: The Action of Cilia and the Effect of Drugs on Their Activity, J. Laryng. & Otol. **49**:571, 1934.

16. Fabricant, N. D.: p_H of Nasal Secretions in Situ in Atrophic Rhinitis, Arch. Otolaryng. **39**:474 (June) 1944.

17. Van Alyea, O. E.: Treatment of Acute Infections of the Nose and Sinuses, Kentucky M. J. **40**:441, 1942.

18. Freund, E. M.: Local Use of Some of the Newer Sulfonamide Solutions in Disease of the Ear, Nose and Throat, Eye, Ear, Nose & Throat Monthly **23**: 104, 1944.

19. Crowe, S. J., and Ward, A. T., Jr.: Local Use of Sulfadiazine, Tyrothricin, Penicillin and Radon in Otolaryngology, North Carolina M. J. **4**:431, 1943.

and George,[20] Livingston,[21] Bordley, Crowe, Dolowitz and Pickrell[22]; Harris, Sommer and Chapple,[23] and Sulman[24]). O'Donnell[25] reported 2 cases in which Paredrine-sulfathiazole suspension produced toxic reactions.

In going over the literature, I could find no articles in which these more recent combinations of drugs had been tried on animals to see the effect on the nasal mucosa, as had been done with sodium sulfathiazole sesquihydrate. In a personal communication from Dr. Charles F. Church, he said that in his laboratory Sulmefrin (sodium sulfathiazole and desoxyephedrine) had been sprayed three times a day in rabbits for ten days, with no ill effects to the nasal mucosa.[26]

The present paper describes the results of the use on rabbits of six of the sulfonamide combinations now on the market. Fifteen rabbits were used. In one group the drugs were used for five days, and in the other group they were instilled for fifteen days. Three rabbits served as controls: 1 with isotonic solution of sodium chloride, 1 with 5 per cent sodium sulfathiazole and 1 with no drug used (table 1).

At the end of the experimental period, the rabbits were killed. The heads were removed and split longitudinally. The septums and maxilloturbinal and ethmoturbinal bones were dissected out. Sections were made of these bones with their mucous membrane and examined. The results, grossly and microscopically, are shown in table 2.

Experiment 1 was repeated on 4 rabbits. The rabbits were killed and sections made as in the other experiments. These sections all showed normal mucosa. Thus it was concluded that the damage found in the mucosa of the first rabbit was due to other factors.

The experiments indicate that the mucosa of rabbits was not injured by the various drugs and that it was damaged by 5 per cent sodium

20. Turnbull, F. M.; Hamilton, W. F.; Simon, E., and George, M. F., Jr.: Sinusitis and Infections Secondary to the Common Colds, J. A. M. A. **123**:536 (Oct. 30) 1943.

21. Livingston, G. S.: Clinical Experience with Sulfonamides in Otolaryngology, Ann. Otol., Rhin. & Laryng. **52**:171, 1943.

22. Bordley, J. E.; Crowe, S. J.; Dolowitz, D. A., and Pickrell, K. L.: The Local Use of Sulfonamides, Gramicidin and Penicillin in Otolaryngology, Ann. Otol., Rhin. & Laryng. **51**:936, 1942.

23. Harris, T. N.; Sommer, H. E., and Chapple, C. C.: The Administration of Sulfonamide Micro-Crystals by Inhalation, Am. J. M. Sc. **205**:1, 1943.

24. Sulman, L. D.: Paredrine Hydrobromide-Sulfathiazole Therapy of Infections of the Upper Respiratory Tract, Arch. Otolaryng. **37**:713 (May) 1943.

25. O'Donnell, M. C.: Toxic Reactions of Paredrine-Sulfathiazole Suspension, J. A. M. A. **122**:298 (May 29) 1943.

26. Church, C. F.: Personal communication to the author.

TABLE 1.—*p_H of Drugs Given by Drug House and p_H at End of Experiment*

Experiment	Name of Drug	Days Instilled	p_H Given by Drug House	p_H at End of Experiment
1	Thizodrin	5	8.5 - 9.0	9.2
2	Sulfadrine	5	5.5 - 6.5	5.4
3	Gluco-Fedrin with sulfathiazole	5	5.4 - 5.8	5.4
4	Sulfedex	5	9.1	9.1
5	Paredrine-sulfathiazole suspension	5	5.5 - 6.5	6.55
6	Sulmefrin	5	9	9.4
7	Thizodrin	15	8.5 - 9.0	9.2
8	Sulfadrine	15	5.5 - 6.5	5.4
9	Gluco-Fedrin with sulfathiazole	15	5.4 - 5.8	5
10	Sulfedex	15	9.1	9.1
11	Paredrine-sulfathiazole suspension	15	5.5 - 6.5	6.55
12	Sulmefrin	15	9	9.4
13	5 per cent sodium sulfathiazole sesquihydrate (control)	15	.....	9.5
14	Isotonic solution of sodium chloride (control)	15	.....	7.35
15	No drug (control)	15		

TABLE 2.—*Results of Instillation of Sulfonamide Drugs in Rabbits*

Experiment	Drug	Days	Gross Findings	Microscopic Observations
1	Thizodrin	5	Normal	Desquamation of mucosa; collection of acute purulent exudate in crypts; experiment repeated on 4 rabbits; sections made showed normal epithelium
2	Sulfadrine	5	Normal	Normal
3	Gluco-Fedrin with sulfathiazole suspension	5	Normal	Normal
4	Sulfedex	5	Normal	Normal
5	Paredrine-sulfathiazole suspension	5	Normal	Normal
6	Sulmefrin	5	Normal	Normal
7	Thizodrin	15	Normal	Normal
8	Sulfadrine	15	Few pus cells in folds of turbinates	Normal
9	Gluco-Fedrin with sulfathiazole suspension	15	Few pus cells in folds of turbinates	Normal
10	Sulfedex	15	Normal	Normal
11	Paredrine-sulfathiazole suspension	15	Membrane hemorrhagic	Normal
12	Sulmefrin	15	Normal	Normal
13	5 per cent sodium sulfathiazole	15	Membrane hemorrhagic; pus found in turbinates	Necrosis of mucous membrane; surface epithelium partially sloughed off
14	0.8 per cent sodium chloride	15	Normal	Few polymorphonuclear cells in crypts
15	No drug (control)	15	Normal	Normal

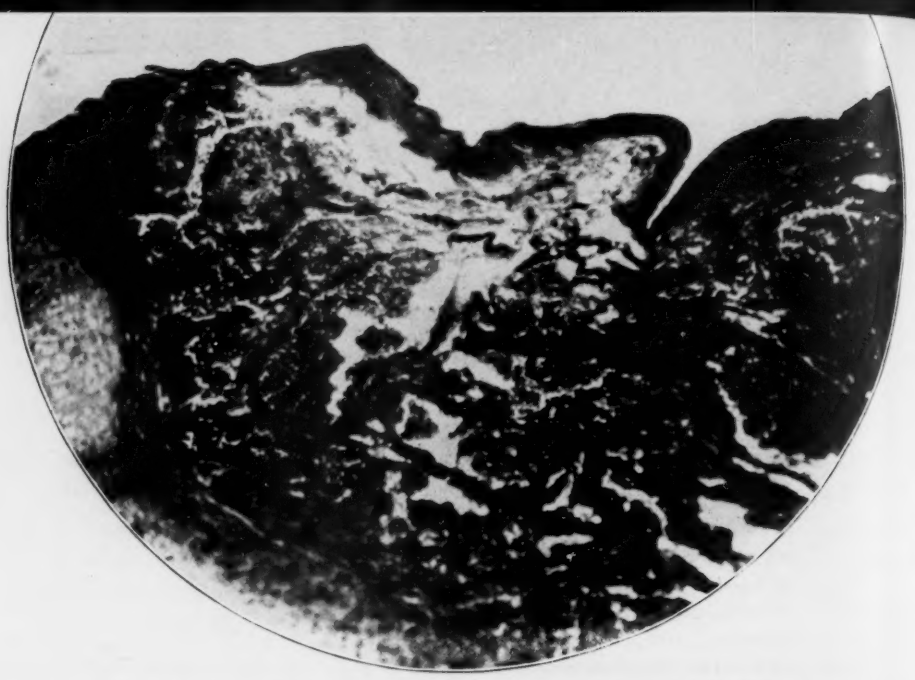


Fig. 1.—Section of epithelium of rabbit in which Gluco-Fedrin with sulfathiazole was used. This shows a normal, undamaged mucous membrane. This section is typical of the epithelium of the rabbits which had the six sulfonamide drugs instilled for five or fifteen days.

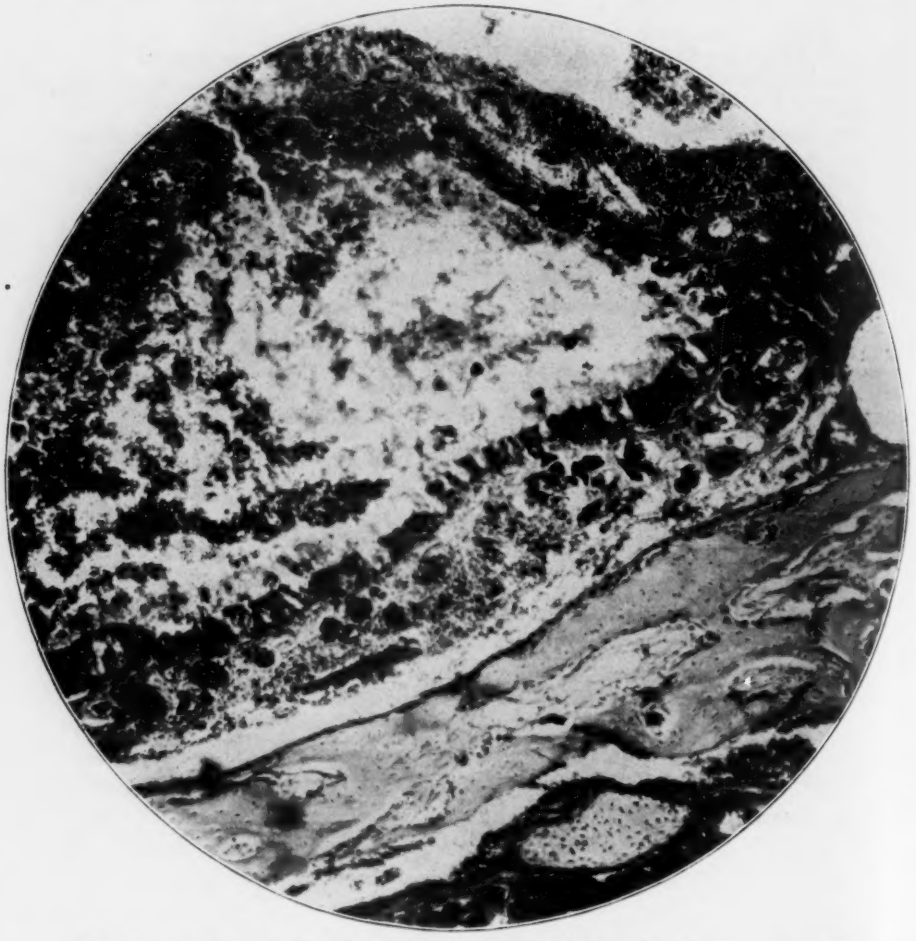


Fig. 2.—Section of the epithelium from the rabbit with 5 per cent sodium sulfathiazole for fifteen days. This shows pronounced superficial necrosis of the mucous membrane, with collection of pus on the surface. In places the membrane has sloughed away. In submucosa there is edema, swelling, dilatation of the capillaries and a collection of polymorphonuclear leukocytes.

sulfathiazole (figs. 1, 2 and 3). The p_H range was considerable, 5.0 to 9.4, in the six drugs. However, no ill effects was found from those with a low p_H or a high p_H.

CONCLUSIONS

1. The six drugs used, Thizodrin, Sulfadrine, Gluco-Fedrin with sulfathiazole, Sulfedex, paredrine-sulfathiazole suspension and Sulmefrin, produced no ill effects in the mucous membrane of rabbits.

Fig. 3.—Section of epithelium of the rabbit given 0.9 per cent sodium chloride for fifteen days. This shows a normal mucous membrane with a few polymorphonuclear leukocytes on the surface and in the submucosa.

2. Since it is generally accepted that the mucosa of rabbits is similar to that of human beings, these drugs do not injure the epithelium of the nose.

3. The nasal mucous membrane, since the p_H of the various nose drops varied from 5 to 9.4, can apparently stand a considerable change of p_H.

Dr. S. A. Wallace and Dr. Paul A. Wheeler, of the Department of Pathology of Baylor University College of Medicine, offered suggestions, aid and help of great value in this work.

Case Reports

BIFID NOSE ASSOCIATED WITH MIDLINE CLEFT OF THE UPPER LIP

D. F. WEAVER, M.D., and D. H. BELLINGER, D.D.S., DETROIT

A THOROUGH examination of the medical literature reveals only 13 cases reported dealing with the deformity variously known as bifid nose (2 cases),[1] median fissure (4 cases),[2] cleft nose (1 case),[3] median furrow (1 case),[4] double nose (3 cases)[5] and nose with four nostrils (1 case).[6] There was only 1 case of bifid nose associated with midline cleft of the upper lip.[7] Only three of the reports were made in the North American literature.[8]

Kopp's [1a] report dealt with a bifid nose associated with a deformity of the frontonasal bony framework.

Wilkinson [1b] reported a case of bifid nose and stated:

> From the mesial (nasal) processes are developed the nasal septum, the columella, the mid-portion of the (upper) lip and the pre-maxilla. These processes may fail to unite in the second month. True median hare lip is the best known deformity which may result. Other deformities referable to the same cause are median cleft palate, bifid nose and median dermoid cysts or fistulae of the ridge of the nose.

1. (*a*) Kopp, M. M.: Two Congenital Deformities: Bifid Nose and Bulldog Nose, Laryngoscope **49**:1128-1133, 1939. (*b*) Wilkinson, G.: Case of Bifid Nose, J. Laryng. **37**:560-563, 1922.

2. (*a*) Esser, E.: Median Fissure: Surgical Therapy of Cases, Plastic Chir. **1**:40-50, 1939. (*b*) Thomas, L.: Observation de fissure congenitale du nez, Bull. Soc. de chir. de Paris **2**:162-167, 1873. (*c*) Poland, J.: Congenital Median Fissure of the Nose, Pediatrics **1**:401, 1896. (*d*) Zeno, L.: Median Fissure with Hypertelorism: Case, An. de cir. **8**:37-39, 1942.

3. Eitner, E.: Correction of Cleft Nose, Deutsche Ztschr. f. Chir. **252**:507-510, 1939.

4. Goodale, J. L.: A Case of Nasal Deformity from a Median Furrow, Corrected by Subcutaneous Implantation of a Portion of the Septal Cartilage, Boston M. & S. J. **145**:93, 1901.

5. Regnault, F.: Le femme a deux nez et le polyzoisme teratologique, Bull. et mém. Soc. d'anthrop. **2**:333-337, 1901. Baumgarten, E.: Nez anterieur double: Deux cloisons cartilagineuses et trois narines, Rev. de laryng. **14**:10-12, 1894. Muecke, F. F., and Souttar, H. S.: Case of Double Nose, Proc. Roy. Soc. Med. (Sect. Laryng.) **17**:8, 1923-1924.

6. Thomson, St. C.: Congenital Deformity Showing a Nose with Four Nostrils, J. Laryng. **24**:207, 1919.

7. Lagos, G. A.: Median Harelip and Bifid Nose: Case, Semana méd. **2**:237-241, 1944.

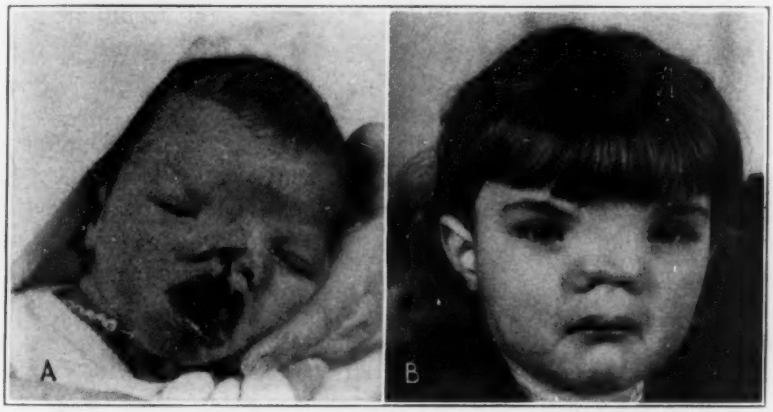
8. Kopp.[1a] Poland.[2c] Goodale.[4]

According to Arey,[9] the side and wing of the nose as well as the frontal processes of the superior maxilla are derived from the lateral nasal process, and the forehead is derived from the frontal process.

In the following case, in addition to the bifid nose and midline cleft of the upper lip, there is excessive width between the nasal processes of the superior maxilla and also between the eyes. This would indicate that the deformity is a result not only of failure of the mesial nasal processes to unite but also of anomalous development of the lateral nasal processes.

REPORT OF A CASE

A white female baby was born at full term at the Henry Ford Hospital on March 31, 1943. Her physical condition was normal in every way except for a bifid nose associated with a midline cleft of the upper lip (figure, *A*). One normal child had been born previously to the mother.



A, appearance of infant at birth. *B*, appearance of child at present.

On April 13, 1943, with the infant under anesthesia carried out by drop ether, Dr. Bellinger repaired the cleft lip. The mucous membrane was approximated with fine interrupted silk sutures and the skin with interrupted sutures of Kaldermic.

On June 31, 1943, with the infant under anesthesia carried out by drop ether, Dr. Weaver repaired the cleft nose. Incisions were made which extended along the margin of the upper half of each nostril and met at a point on the upper lip which was to be the location of the base of the columella. A wide area of skin was undermined over the nasal region and cheeks, including the skin over the alar cartilages. The nasal septum was found to be widely divided in the midline, and each half was attached to its corresponding alar cartilage. The alar cartilages were then brought together by a through and through silk suture, which was tied over a small rubber tube at each nasolabial sulcus. Several more through and through silk sutures were used to approximate the two halves of the columella,

9. Arey, L. B.: Developmental Anatomy, Philadelphia, W. B. Saunders Company, 1942.

the two halves of the septal cartilage and the two alar cartilages. These were tied over small cotton rolls. The excess skin was excised, a suitable flap being left to cover the anterior surface of the columella. The cutaneous edge was then sutured to the margin of the upper half of the nostril and the columella with fine interrupted silk sutures. A congenital pit in the skin at the junction of the upper surface of the alar cartilages was left undisturbed. No attempt was made to approximate the nasal processes of the superior maxilla.

The postoperative course was uneventful. The airway was adequate on both sides.

On July 3, 1944, with the patient under anesthesia carried out by drop ether, the congenital pit in the skin, in which some hair had developed, was excised and the upper edges of the alar cartilages approximated more closely. The skin was closed with interrupted sutures of fine silk.

The postoperative course was uneventful (figure, *B*). Roentgenograms of the sinuses show normal development.

COMMENT

At present the width of the nasal bridge is excessive, as is the distance between the two eyes. The measurement from one inner canthus to the other is 4.5 cm. Her mother's measurement for this distance is 3 cm. and her father's 3.25 cm.

Due consideration has been given to narrowing of the nasal bridge, but it is felt that approximation of the nasal processes of the superior maxilla cannot be accurately carried out until proper development has taken place. What effect the attempt would have on proper development of the sinuses and lacrimal ducts is uncertain. For these reasons and because the eyes are too far apart, it is felt that the final result will be better if nothing further is done until the development is complete. Then, if thought advisable, the nasal bridge can be narrowed by refracturing the nasal processes of the superior maxilla, and a suitable cartilage graft can be inserted.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

FUNCTONAL EXAMINATION OF HEARING

ALFRED LEWY, M.D., and S. L. SHAPIRO, M.D.
CHICAGO

EUROPEAN literature on otolaryngology, except that from Switzerland, is still scant. To some extent this is made up by an increased output from South American countries. A considerable proportion of the articles on the subject are concerned with traumatic deafness both in war and industry, its prevention and rehabilitation measures. There is a large increase in the number of acceptable hearing aids, and there is a noticeable effort to produce satisfactory ones at a lower price than formerly. A few keen investigations of the physiology of hearing are very encouraging.

HEARING AIDS

In an instructive article, L. A. Watson [1] discusses the prescribing of hearing aids from the viewpoint of the acoustic engineer. It is his contention that the audiometer offers the most reliable method for analyzing hearing impairments and prescribing and fitting hearing aids. Spoken voice and intelligibility tests for this purpose are unsuitable because of certain inherent sources of error. Chief among these are variation in the sound power of different voices, modifying effects on the voice of the acoustic properties in the test room, variations in the sound power of different speech elements and differences in enunciation on the part of the speaker as well as differences in linguistic aptitude in the subject.

A short list of words or sentences is misleading as a test of hearing because it does not allow for the factors of effort and fatigue which may not become manifest until an attempt is made to use the hearing aid for a longer period. There is no objection to using speech intelligibility tests as an adjunct to the pure tone audiometric tests, but the latter are the only means for separating and measuring the many constituent elements of hearing correctly. To reveal the fatigue element in a badly fitting aid, three or more hours are needed of successive ten minute tests with both sentences and random words.

The question of who needs a hearing aid is to be determined, according to Watson, on the basis of percentage of hearing loss as decided

1. Watson, L. A.: Certain Fundamental Principles in Prescribing and Fitting Hearing Aids, Laryngoscope **54**:531 (Oct.) 1944.

either from the table of the American Medical Association or from the simpler method of Fowler. Using the first table, a loss of 25 per cent in the better ear indicates need for a hearing aid, while Fowler's schedule

Hearing Aids Accepted by the Council on Physical Medicine of the American Medical Association

Hearing Aid and Manufacturer	Issue of J. A. M. A. Containing Report
Aurex (Semi-Portable) (tube)	109 : 585 (Aug. 21) 1937
Aurex Model C-B and Model C-A (tube)	120 : 535 (Oct. 17) 1942
Aurex Corp., 1117 N. Franklin St., Chicago	
Beltone Model 603H (tube)	124 : 1059 (April 8) 1944
Beltone Hearing Aid Co., 847 W. Jackson Blvd., Chicago	
Gem Model V-4, C-186 (tube)	120 : 1397 (Dec. 26) 1942
Gem Ear Phone Co., 47 W. 34th St., New York	
Maico Model 42 (tube)	120 : 839 (Nov. 14) 1942
Maico Ace (tube)	121 : 48 (Jan. 2) 1943
Maico Type K	Report to be published
Maico Company, Inc., 2632-36 Nicollet Ave., Minneapolis	
Mears Aurophone, No. 98 (tube)	118 : 978 (March 21) 1942
Mears Radio Hearing Device Corp., 1 West 34th St., New York	
Otarion, Model A-1 (tube)	115 : 1101 (Sept. 28) 1940
Otarion, Model A-2 (tube)	125 : 849 (July 22) 1944
Otarion, Inc., 448 N. Wells St., Chicago	
Paravox Models VV2 and VV3 (tube)	128 : 879 (July 12) 1945
Paraphone Hearing Aid Co., 2056 E. 4th St., Cleveland 15	
Radioear, Model B-20 (carbon)	110 : 1753 (May 21) 1938
Radioear Masterpiece (carbon)	117 : 2169 (Dec. 20) 1941
Radioear Model 45M—Magnetic bone conduction receiver (tube)	127 : 27 (Jan. 6) 1945
Radioear Model 45M—Magnetic air conduction receiver (tube)	127 : 219 (Jan. 27) 1945
Radioear Model 45CM (tube)	126 : 1151 (Dec. 30) 1944
E. A. Myers & Sons, 306-8 Beverly Road, Mt. Lebanon, Pittsburgh	
Sonotone Audicles #530, 531, 533 (tube)	123 : 837 (Nov. 27) 1943
Sonotone Corp., Elmsford, N. Y.	
Telex Model 612 (tube)	114 : 1634 (April 27) 1940
Telex Model 900 (tube)	117 : 1978 (Dec. 6) 1941
Telex Model 1020 (tube)	117 : 2072 (Dec. 13) 1941
Telex Model 1550 (tube)	126 : 705 (Nov. 11) 1944
Telex Products Co., 1645 Hennepin Ave., Minneapolis	
Vacolite Model D (tube)	118 : 896 (March 14) 1942
Vacolite Company, 3003 N. Henderson, Dallas, Tex.	
Western Electric Audiphone Ortho-technic Model (carbon)	112 : 1062 (March 18) 1939
Western Electric Ortho-tronic Audiphone (tube)	121 : 1283 (April 17) 1943
Western Electric Telephone Type Audiphone, Model J-1 (carbon)	125 : 849 (July 22) 1944
Western Electric Company, 300 Central Ave., Kearny, N. J.	
Rovox (Semi-Portable) (tube)	113 : 1643 (Oct. 28) 1939
Zenith Model B-3-A (tube)	127 : 158 (Jan. 20) 1945
Zenith, Model A-3-A (tube)	127 : 159 (Jan. 20) 1945
Zenith, Model A-2-A (tube)	127 : 159 (Jan. 20) 1945
Zenith Radionic Model HA (tube)	124 : 361 (Feb. 5) 1944
Zenith Radio Corp., 6001 Dickens Ave., Chicago	
Maico Audiometer Model D-5	114 : 139 (Jan. 13) 1940
Maico Audiometer Model D-8	120 : 205 (Sept. 19) 1942
Maico Company, Inc., 2632-36 Nicollet Ave., Minneapolis	
Sonotone Audiometer, Model 20	124 : 94 (Jan. 8) 1944
Sonotone Corporation, Elmsford, N. Y.	
Western Electric Audiometer Model 6B	114 : 1634 (April 27) 1940
Western Electric 4C Audiometer	118 : 1297 (April 11) 1942
Western Electric Company, 300 Central Ave., Kearny, N. J.	

calls for an aid when the loss is between 30 and 40 per cent. The area between the 30 and 40 per cent lines on the audiogram is the critical area, and Watson correctly stresses the importance of recruitment in determining whether or not an aid is needed before all this area is lost.

In discussing the problem of who can be benefited by a hearing aid, Watson makes the statement that many persons who are hard of hearing, with losses of 90 decibels at 1024 double vibrations and 95 decibels at 2048, are able to carry on direct conversation and can understand normal speech fairly well at distances of 5 to 10 feet (152.4 to 304.8 cm.) or more if they are equipped with the best modern aid. Losses greater than this are unsuitable for hearing aids. Unusual curves, such as the U-shaped curve, or audiograms which end sharply at 1024 involve interpretative difficulties which are difficult to correct with hearing aids. Audiograms to illustrate the points made are presented.

The principles for fitting hearing aids are outlined as follows:

1. If the better ear has a loss of less than 50 db. in the most essential tones of the speech range (512 to 2,896), fit the poorer ear if good results can be obtained on that ear.

2. If the better ear has a loss of more than 50 db. in the essential speech range, the better ear may be fitted because it is of almost no direct benefit without a hearing aid and no binaural advantage will be obtained through fitting the poorer ear.

3. If the poorer ear has a severe loss of over 75 db. or has a limited frequency range, or exhibits very poor ability to interpret and understand speech, the better ear may be fitted even though the loss on the better ear is less than 50 db.

4. If the threshold curves of the two ears are nearly identical, it is well to fit the ear which has the best perceptive and interpretative ability. The factor of recruitment is also important. Recruitment at lower intensity levels is an advantage to an ear. An ear with 50 db. or less of loss which has a strong initial recruitment just above threshold should be left unfitted and the other ear with less recruitment fitted.

Bone conduction receivers, which were popular for carbon aids, have only limited use with the vacuum type of instrument. This is because while emphasizing the lower and middle frequency ranges they give little or no reproduction of sounds above 2000 cycles. Since they require considerably greater power than the usual air conduction receiver, bone conduction should be used only when there is an increase of at least 30 decibels in the bone conduction curve over the air conduction curve in the frequency range between 512 and 2896 cycles.

The author stresses the great importance of the factor of recruitment in determining the ease or difficulty with which the patient can be fitted. Thus a patient with a loss at threshold of 50 decibels requiring amplification to 90 decibels for conversation will hear these sounds with the same intensity that a normal person would hear a sound of 40 decibels if no recruitment is present; if recruitment exists, however, the sound would be heard at an intensity of 90 decibels, which is too loud for comfort. Watson advises measuring the range of comfortable loudness in addition to the usual threshold determination; this involves measuring both pure tones and speech through a microphone first at that level at

which the sound is loud enough to be easily audible and yet not uncomfortably loud, and afterward at that point at which the sound becomes painful to the subject. The area between the two levels is the range of comfortable loudness, which may not be exceeded if the ear is to be fitted successfully.

Watson lists the different ways in which manufacturers of hearing aids provide amplification and frequency variations to suit different types of deafness with and without recruitment. These include different types of transmitters, tone controls, switch controls, changes in the receiver, changes in microphones and changes in frequency by set screw adjustments. Thus there is an opportunity for many different combinations if all methods are used. Illustrative curves are presented.

The role of the otologist is seen to be that of adviser and judge of the merits of the various instruments offered for sale. It is essential that the physician convince his patient that a hearing aid is no quick and easy miracle, but an instrument which requires adjustment and practice, because otherwise the psychologic resistance to hearing aids which is present in so many persons will lead them to discard the aid without giving it a fair chance to demonstrate its usefulness. While Hughson and some others believe that the physician should make the impression from which the individually molded earpiece is produced, actually most otologists find this job too tedious and refuse to bother with it. The very least, however, that they can do is to study the external canal and correct any condition which may interfere with making the impression or cause discomfort to the patient. Watson does not recommend the wide channel ear insert favored by Grossman [2] because it may increase the feedback problem. Mention is made of a new type of plastic impression material which eliminates the hazards of dental impression plaster; it does not adhere to the walls of the canal and is easy to remove.

The investigations are described in detail and present evidence of errors which occur in present methods of audiometric testing of hearing by bone conduction. They are in the bone conduction itself and not due to the manner of testing or to psychologic factors.

HEARING TESTS

J. Kent Leasure [3] has devised a hearing testing cabinet suitable for office installation. The height is 7 feet (213.4 cm.), length 6 feet (182.9 cm.), width 3 feet (44 cm.). The walls and top are made of

2. Grossman, F. M., and Molloy, C. T.: Physical Characteristics of Some Bone Oscillators Used with Commercially Available Audiometers, Arch. Otolaryng. **40**:282 (Oct.) 1944.

3. Leasure, J. K.: Hearing Testing Cabinet, Ann. Otol., Rhin. & Laryng. **54**:183 (March) 1945.

½ inch (1.3 cm.) pressed wood pulp; the floor, which is not attached, of ordinary wood flooring. The sides and top are divided into partitions of 3 inch (7.6 cm.) squares, filled with insulating material which does not settle. The doors take up the full height and most of the width, so that when the doors are opened the air is changed quickly. There is room for the patient, physician and one observer, particularly a mother. He uses a light signal independent of the audiometer. While the installation is not technically sound proof, he claims that if rubber padded receivers are used detection of hearing at the zero line is possible, while tests in the average office will not detect losses of less than 20 to 30 decibels.

Tests of Unilateral Deafness.—Robert E. Priest[4] describes his examination routine in claims of total unilateral deafness. After the usual voice, tuning fork and audiogram tests are recorded and repeated to detect inconsistencies, the following are recommended: the shadow curve audiogram as recommended by Bunch; the Wells-Stenger test, a modification in which the stem of the fork is inserted into a rubber tube 30 inches (76.2 cm.) long and with a diameter of ¼ inch (0.64 cm.), thus requiring only one fork for both masking and hearing distance in the good ear [we prefer the original Stenger test when two identical forks are available]; the Lombard test; the Marx test, and a stream of water in lieu of the noise apparatus. As auxiliary tests he recommends the Becker watch and tube test (not applicable when high tones are lost). The watch is pressed against a rubber tube connecting both ears and moved nearer one or the other. The tube may be pinched to cut off the sound from either ear. In the double conversation test two rubber tubes and two speakers at the same time are supposed to confuse the hearer if he hears in both ears. It is possible to concentrate on one ear. In the Becker bone and air conduction test, in which a 30 inch rubber tube is used with a closed end looped around a tuning fork, the other end with an ear piece, the same technic is employed as in the Wells-Stenger test except that both air and bone conduction can be tried. The Teal test is used when the patient admits hearing by bone but denies hearing by air conduction for the tuning fork. Absence of the caloric reaction is supporting evidence of a claim of deafness. The tests are described in detail. The Tschudi, Erhard, Chimani-Moos, Wotzilka lid reflex and Nadoleczny tests were discarded as unreliable or impracticable. The routine test record is appended.

Lieutenant Commander Russell Fletcher,[5] after describing the basis of the Stenger test and a description of its technic with the tuning forks,

4. Priest, R. E.: Tests for Unilateral Deafness, Arch. Otolaryng. **42**:138 (Aug.) 1945.

(Footnotes continued on next page)

describes his method of using the audiometer. Two ear phones of the same calibration as the one ordinarily used for the air conduction test are connected into the air conduction outlet and into the bone conduction outlet respectively. The audiometer is set for a medium frequency. The allegedly deaf ear is tested in the routine way (hearing is denied). The good ear is tested with the other receiver connected with the bone conduction outlet and the dial is turned to the volume that permits hearing at 20 inches (50.8 cm.) (this requires about 50 decibels for a normal ear). The patient is blindfolded and the receiver, connected with the air conduction outlet, is again placed against the allegedly deaf ear (the author does not specify the intensity, but presumably it should be the same as in the other receiver), and the distance is measured at which the tone is heard in the good ear. If the tone is heard in the good ear at the same distance with or without the receiver at the deaf ear, the patient is really deaf, and the finding is recorded as 20/20. If he hears at only 3 inches (7.6 cm.) it is recorded as 3/20, and the patient is shown to be a malingerer. A large ordinary room is preferable to a small soundproof one, which acts as a sounding board when both receivers are operating at high intensity.

Tests for Detecting Simulated Bilateral Deafness.—Escalada [6] deals with tests for detecting simulated deafness. The article is written for Argentine army physicians and necessarily stresses those tests which do not require special otologic training.

For simulated bilateral deafness, time-honored simple strategems such as having an assistant casually remark to the patient, "Your pants are open," while the physician is apparently busy with something other than examining the patient, are quoted with approval. Sudden noises behind the patient, unexpected questions or statements, such as "You have forgotten the date" after the subject has been asked to write something, even a pretended aside to the assistant on the necessity of opening the drum while in the act of examining the ear through a speculum are devices used by the author.

Barth's test depends on the fact that a person with bilateral deafness cannot sing in tune. The suspect is asked to write a song with which he is familiar, singing it at the same time. To distract the attention the following device is used: The man is asked to read the letters on a visual chart as they are pointed out with the finger; at the appropriate moment, in place of signaling the letters the subject is told, "Read the second line," or "Read the red letters." The author recommends highly

5. Fletcher, R.: The Stenger Malingering Test Made with the Audiometer, Ann. Otol., Rhin. & Laryng. **54**:362 (June) 1945.

6. Escalada, O. O.: Simulation of Deafness, Rev. san. mil., Buenos Aires **43**:1652 (Dec.) 1944.

the device of Oudot. In this a patient with unilateral deafness is placed with the "deaf" ear toward the examiner while the other is firmly occluded with the finger. Standing at a distance of 5 meters the physician says, "When you hear me raise the right arm." Most simulators will raise the arm when the examiner reaches a distance of 0.5 meters; the fact that they execute the command proves that they heard at 5 meters. In the Nadoleczny test the patient first pronounces aloud simple numerals which he reads from the lips of the examiner who makes no actual sound; during this procedure an assistant whispers different numerals in the "deaf" ear; there will be no hesitation or difficulty on the part of the patient in repeating the numbers he reads from the examiner's lips if real deafness is present. The method of Helsmoortel and Nyssen of studying the cochleovascular reactions to sound by means of Marey's drum and a recording needle is also given. The Stenger test and its variations are given as well as those which show bad faith, such as denying hearing a fork of 4096 double vibrations before the bad ear (high tones carry around to the other ear) while the "good" ear is not occluded, the false Weber (patient hears nothing by bone conduction on the vertex when the good ear is occluded) and the tests of Escat and Tschudi are given in detail. The author remarks that the examiner must be something of a psychologist and adjust his tests in accordance with the mentality and education of the patient.

Speech Tests.—In a comprehensive article on speech tests, Douglas MacFarlan [7] gives a historical and up to date survey of the development and progress of this phase of functional testing. It has long been realized that speech is the most important thing with which hearing is concerned, and the advent of the pure tone audiometer has not changed this concept, since it is difficult to establish an accurate relationship between the audiograms and the ability to hear the spoken voice. As long ago as 1904 W. S. Bryant reported on a phonograph "acumeter" in which air-borne words were carried from a phonograph record by means of stethoscope tubes, with a device for increasing the intensity of the sound. An electric phonograph with a pick-up that activated a telephone circuit and a rheostat for controlling intensity was devised by Professor Bristol for the Lexington Avenue School for the Deaf, the list of words appropriate for children being supplied by Miss Berry of that institution.

Dayton Miller, Crandell and, above all, Fletcher did exhaustive work on the frequencies of vowels and consonants. The Western Electric 4-A audiometer came out in the later twenties and attained great popularity for testing school children; three digit numbers for older children and two digit numbers for younger ones with attenuation in three decibel steps

7. MacFarlan, D.: Speech Hearing Tests, Laryngoscope **55**:71 (Feb.) 1945.

were used. MacFarlan considers the method crude but simple and effective in spite of certain faults, as it caught many cases of unsuspected deafness.

A good summary and comment are given of research by the Bell Laboratory staff in connection with testing telephone circuits. MacFarlan's opinion is that while much useful and even practical knowledge was achieved the articulation lists of meaningless sounds (or nonsense syllables) are of little value to the otologist.

In speech testing, inflection, accent, dialect and other characteristics other than the physical attributes of the sound are of importance. Because of their familiarity numbers give a 10 per cent better hearing performance than other words. The author prefers a constant level record for number tests. A lower threshold can be achieved when going from audible to inaudible intensity than in coming from an inaudible level to audible. Practical suggestions on carrying out speech tests with phonograph records are given.

A full list of the recent word selections including the author's own are given, as well as some excellent previously unpublished selections for children of various ages by Leila Larsen of Indiana University. The selection of Wegel and the word list by Dr. Robert West of the University of Wisconsin are fully given, as well as the list and directions for use worked out by Watson and Knudsen and the tables by Miss Kerridge of London. A description is given of a speech audiometer by L. Clyde Dwight, with a record embodying groups of words of selected frequencies and a summary is given of an excellent unpublished treatise on speech-hearing testing by Alfred R. Thea. Sentence intelligibility lists, such as those reported by the Bell Laboratories, are quoted also. MacFarlan discusses the fallacy of attempting to determine how well speech is heard from an audiogram and cites a number of audiograms and results of Western Electric 4-A audiometer tests, comparing the findings in each instance to bring out his point.

The author has made an admirable contribution in summarizing present day methods and basis for speech-hearing tests, but has not sufficiently stressed the technical difficulties which remain to be overcome before these tests can be made accurately by the average otologist. While it is true, as MacFarlan states, that the quality of acoustic circuits has reached a point of remarkable perfection and fidelity and the decibel meter has made it possible accurately to measure loudness of speech, testing with a microphone and oscillograph are hardly likely, for obvious reasons, to be adapted to everyday practice. On the other hand, the phonograph record, aside from the question of wear, is so subject to variation as to type of voice, method of recording and above all surface noise, that standardization thus far cannot be even considered

It is to be hoped that some of the new methods of recording, such as that with steel wire, can remove these objections so that fine work done in the field of speech-hearing tests can find practical application on a large scale.

Hearing Tests for Young Children.—An article on hearing tests for young children by I. R. Ewing and A. W. G. Ewing [8] discusses the problem mainly from the educational and psychologic viewpoint. The study was conducted by the department of Education of the Deaf, University of Manchester, on children attending War Nurseries and babies in the Maternity and Child Welfare Centers; 91 children and babies were classified as to age group and response.

The results are given in considerable detail and cannot be given here in full. In general it was found that standardizing the tests or timing them was useless because the interests and behavior of children, particularly those under 5, are both variable and individual. Three groups of tests were used: (*a*) voice and speech tests; (*b*) percussion toys including bell, drum, triangle, etc.; (*c*) a range of pitchpipes from 120 cycles to 1900 cycles, and (*d*) "meaningful sounds," e. g., clink of feeding bottle, knock at the door, light tapping, crackling or rustling of paper. Tests were made from a distance of about 6 feet and when the child was not watching. It was found better, except for children in arms, to observe responses when 2 children were together, rather than to make individual tests. For the older children, when more complex voice and auditory discrimination tests were used, a preliminary training period was necessary in most cases.

Some of their conclusions were as follows:

During the first, second and third months of life percussion sounds yield quicker response than voice. As a rule a baby's response is reflex, such as blinking, involuntary jumping, twitching the fingers, etc. During the third, fourth and fifth months voice steadily gains over percussion sounds as a form of stimulation in winning quick response. "Learned" rather than reflex response, indicated by turning toward the source of the sound, begins to be manifest at this age; during the second half of the first year reflexes give way to learned responses, and skill in distinguishing different complex sounds is considerable by the end of this period.

For most children over 1.6 years simple speech tests of hearing are suitable, but children of this age do not pay attention to whispered speech; quiet rather than loud speech wins the interest and attention at this age. Loud percussion and other noises are increasingly ignored. Pitchpipes are not an accurate means of measuring the frequency range

8. Ewing, I. R., and Ewing, A. W. G.: The Ascertainment of Deafness in Infancy and Early Childhood, J. Laryng. & Otol. **59**:309 (Sept.) 1944.

at this age, but are suitable for testing a child between 2 and 3 years of age.

At 3 to 4.11 years whispered voice becomes a suitable means of testing hearing, but pure tone audiometric tests are unsuitable because reliable threshold readings cannot be obtained before the child's interest and attention flag.

In testing preschool children suspected of deafness their studies indicated: If a child does not respond to the sound of voice by the end of the first year he is either deaf or has some other form of gross defect. If it is shown that a child never turns his eyes or head toward a familiar person calling his name in a moderately strong voice he is likely to be totally or severely deaf. In children aged 1.0 to 1.11 years whose hearing is impaired, reflex response is of much more frequent occurrence than with ordinary children of this age, and ability to locate the sound the first time it was heard is absent. During the third year voice tests were found to be suitable for partially deaf children, and pitchpipes were useful for testing the frequency range. No capacity to locate sound was present. With children up to 4.11 years more complex tests using various degrees of loudness of voice with vowels and consonants were found suitable after a preliminary training period; children who failed to follow speech were given voice tests of a simpler nature. Audiometric tests were found to be unsuitable.

CONDUCTION DEAFNESS

Conduction Deafness.—The usual concept that conduction deafness is characterized by loss of low tones while nerve deafness involves mainly loss of high tones is criticized by Gurria Urgell.[9] The author cites previous conclusions in other communications on otologic tests which he has presented before the National Academy of Medicine. These were to the effect that there are two methods of transmission for sound, the mechanical, which begins in the external auditory canal and ends in the endolymph, and the nervous, beginning with the cochlea and terminating in the cortex. Therefore the present concept which divides impairment of hearing into the stopping of conduction with the stapes and the beginning of perception with the perilymph has no physiologic basis.

The use of the terms "high" and "low" for various tones is also open to criticism, since there is no accord on this point. The terms should not be used without stating whether the tones are to be considered high or low in relation to another given frequency.

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9. Gurria Urgell, D.: Discussion of a Rule in Clinical Otology, Arch. méd. mex. **6**:303 (June) 1944.

The present analysis is confined to the external auditory meatus, and for this purpose the author considers the traditional concept as involving loss of low tones and preservation of high tones in conduction deafness involving lesions not beyond the stapes. Gurria Urgell was led to his study by an observation he noted on reading of the work performed by van Gilse with the method utilized by Wever and Bray,[9a] when he first began to doubt the accuracy of the usual rules for conduction deafness. Van Gilse stated, "On the normal side (left) the external canal was closed with wax and then the fork of 2048 double vibrations was not heard or registered with the galvanometer. The same happened when the left acoustic nerve was cut." This, Gurria Urgell states, indicates that, contrary to what might be expected, the results of a mechanical and of a nerve involvement were the same.

The literature (Fowler, Jones and Knudsen, Pohlman and Kranz and Dowling and Wegel) offers conflicting opinions on the effect of obstructive lesions relative to hearing different frequencies. To resolve these doubts in his own mind, the author conducted experiments in completely and partly obstructing the external canal in normal subjects and testing the hearing before and after removing the obstructing tampon; audiograms of both air and bone conduction are presented. From his studies, Gurria Urgell concludes that the rule referred to at the beginning of the article lacks validity, at least with regard to obstruction of the external auditory canal.

Correa Netto [10] criticizes the technic of the Weber test as commonly employed because in many cases when no lateralization is noted with the usual method of placing a tuning fork of 128 double vibrations on the vertex, lateralization will occur when tested for by the author's method. This consists of placing a fork of 435 double vibrations alternately on each mastoid, when the sound will be found to lateralize in spite of a negative Weber reaction according to the vertex method. For instance, a patient with a chronic otitis media on the right side when tested with a fork of 128 double vibrations on the vertex could not detect any lateralization, but when the tuning fork was placed on the mastoid of the normal left ear he stated that the sound was heard on the right side. This method, complementary to the usual Weber test, Netto calls the method of opposite lateralization. He has verified this test in many instances and chose a fork of 435 double vibrations because it is in the middle of the musical scale. He finds that while in involvement of the inner ear the sound is heard in the better ear, cerebral

9a. van Gilse, P. H. G.: Expérimentations suivant la méthode de Wever et Bray après lésion de la cochlée, Acta oto-laryng. **20**:23, 1934.

10. Correa Netto, O.: The Best Technic for the Weber Test in Examination of Hearing by the Method of Complementary Opposite Lateralization, São Paulo méd. **1**:331 (June) 1944.

lesions, contrary to Frey and Freml, at times cause a Weber reaction on the diseased side. In conditions of the middle ear which nearly always cause a Weber reaction on the diseased side, the occurrence of lateralization to the better ear indicates that the process on the diseased side has passed beyond the confines of the middle ear.

Recruitment.—A discussion of recruitment by Bustamente Gurria and Garibay [11] of the otorhinolaryngology service at the National Institute of Cardiology of Mexico endeavors to classify the types of auditory lesions which may display this phenomenon and to explain the manner of its occurrence.

The authors noted that medical literature does not contain any explanation of the fact that, as Fowler brought out, in certain types of hearing impairment a tone sounded at a given level of intensity may be heard to be as loud as in a normal ear. The difficulty in explaining this occurrence is doubtless largely due to the incomplete knowledge of the role of the cochlea in the analysis of sound—a matter in which, despite much experimental work, present day physicians are hardly better situated than Helmholz and Rutherford.

According to Fowler, Lorente de Nó bases a physiologic explanation of the recruitment phenomenon on the following facts: To stimulate a neuron it is necessary that various stimuli reach it within a short period of time (less than five hundred microseconds). The frequency of the impulses conducted by a fiber cannot be greater than the characteristic capacity of each nerve fiber (about 800 per second). The capacity of the nerve centers for receiving stimuli is, within certain limits, proportional to the number of afferent impulses received in a unit of time. The authors are not in accord with Fowler's conclusion that when a neuron or groups of neurons are for any reason defective, a sufficiently loud stimulus puts other neurons into activity in such a manner that finally the nerve centers receive as many impulses from the affected side as from a normal ear.

Bustamente Gurria and Garibay believe that association cells in the primary acoustic ganglion are in contact with various neurons, and that a given quantity of energy can be brought to the nucleus either by all or (by a process of multiplication) by a part of the afferent fibers and that thereafter the stimulus behaves as if it were a normal one. They believe that loudness depends on the amplitude of the portion of the basal membrane which has been mobilized in the cochlea. They believe further that various grades of auditory nerve lesions may be classed as follows: (*a*) When all the fibers are partly damaged, a stimulus of low intensity draws no response, but as soon as it reaches a sufficient strength to elicit any response at all the loudness of the

11. Bustamente Gurria, A., and Garibay, A.: A Comment on the Fowler Phenomenon, Prensa méd. mex. **9**:124 (Aug. 15) 1944.

sound will be equal to that in a normal ear. Two subgroups are also distinguished. (*b*) When some fibers are completely destroyed and others normal, the biotic balance (as the authors call the level at which the loudness equals that in a normal ear) occurs abruptly at the moment when the auditory fibers carry to the primary ganglion a stimulus sufficient to cause excitation. (*c*) When some fibers are healthy and some partly damaged a degree of loudness equal to that in a normal ear will be reached gradually through a succession of stimuli of increasing intensity. (*d*) When some fibers are partly damaged and others partly destroyed recruitment will not become manifest.

EVALUATION OF HEARING TESTS

In a study conducted on 500 Navy men, Shilling, Everley and Harris [12] report as to the significance and value of the common clinical test of hearing used in the Navy. These include the spoken voice, the whispered voice the coin click and the watch tick tests as compared with the result of audiometric examination. Audiometers used were the Western Electric 2 A and 6 B with tests conducted under partially sound-deadened conditions. A standard procedure was used. The conclusions drawn from the study are given as follows:

1. The Spoken Voice test was shown to be practically worthless as an index of acuity.

2. The Whispered Voice test was shown to be very effective in screening out that type of ear seriously defective for high tones and . . . more or less defective for low tones; the correlation between extent of low tone audiometric loss and whispered voice score was significant but not high. . . .

3. The Coin Click test as used here produced results very similar to those of the Whispered Voice test, but for several reasons the latter is to be preferred.

4. The Watch Tick test differs from the preceding tests in that low tones are involved only very slightly. The Watch Tick test is fairly satisfactory as a differential test of high tone hearing.

5. None of the clinical tests used showed any tendency to measure low tone hearing independently from high tone hearing.

6. In the conduct of this experiment it was found absolutely essential to standardize the technic of giving the various tests.

Two articles by E. Schlittler, writing from Switzerland, deal with the value of the Bezold-Edelmann tuning forks and methods as compared with audiometry. In the first paper he poses three questions: First, is it possible to make a qualitative diagnosis of deafness with more certainty by means of the audiometer than with tuning forks? Second,

12. Shilling, C. W.; Everley, I. A., and Harris, J. D.: Hearing Tests: An Evaluation, U. S. Nav. M. Bull. **44**:100 (Jan.) 1945.

13. Schlittler, E.: Comparative Evaluation of Functional Test According to Bezold-Edelmann (Use of Tuning Forks) and Audiometric Tests, Pract. oto-rhino-laryng. **4**:318, 1942.

can the hearing for speech be estimated better from the audiogram than from an examination with the tuning fork? Third, is the audiogram of more assistance in ordering a hearing aid than the findings with the tuning fork? His material is based on a series of tests of 228 persons with impaired hearing, examined with the Bezold-Edelmann set as well as with a Sonotone Model 1 audiometer. In addition, he drew on previous clinic experience with the Otoaudion and also on tests made with patients in his private practice with a Western Electric Audiometer.

He found that the audiometer does not reach the lowest tones in the scale as do the tuning forks, that the Weber, Rinne and Schwabach tests cannot be done well on the audiometer and that while bone conduction can be measured quantitatively for high tones with the audiometer, a serious drawback is that the sound can also be heard by the opposite ear. He does not believe that masking devices have solved the problem. Curves are shown (some of them involving "shadow curves" in unilateral deafness) in which the audiograms obviously do not give the true picture as regards the type and amount of deafness.

With respect to estimating the defect for speech, he quotes Langenbeck with approval to the effect that "the threshold for understanding speech is not the same as the threshold for hearing [pure tones]." It is Schlittler's opinion that because of the complexity of speech and various factors involved in conversation neither results with a tuning fork nor audiograms can serve as a sure index for estimating the ability to hear speech.

Schlittler thinks that it is absurd to compare the fitting of hearing aids with that of glasses, as he finds the fitting of hearing aids from audiometric curves to be an uncertain affair and usually has to fall back on the trial and error method. We feel that this conclusion does not apply with equal force in this country where better hearing aids and fitting methods are available.

Schlittler[14] in a second communication studies histologically the temporal bones (bilaterally) of 7 patients whose impairment of hearing had previously been studied with the time-honored methods of Bezold-Edelmann, using the forks and Galton whistles. Of the 14 ears, 6 showed the functional picture of a pure conduction involvement; on microscopic investigation there were without exception signs of damage of the middle ear in the form of perforations of the drums, scars, adhesions and thickening of the mucosa, filling up of the oval or round windows, destruction of the ossicles, transudate and cholesteatoma of the middle ear. Eight ears showed the antemortem picture of perception deafness; in 4 of these the anatomic substratum was a degenera-

14. Schlittler, E.: Functional Findings and Anatomic Substratum, Pract. oto-rhino-laryng. **5**:308, 1943.

tion of the cochlear nerve, disappearance of the ganglion cells or atrophy of the end nerves in the basal turn of the cochlea, while the remaining four temporal bones demonstrated definite changes in the internal ear.

Schlittler states that although this type of investigation has been done before the evidence he presents is added proof of the value of the Bezold-Edelmann methods as a practical means of diagnosing impairment of hearing and that audiometry should only be an adjunct to the proved tests, such as the Weber, Schwabach and Rinne tests.

Fowler [15] has observed 2 patients with monaural total or almost total acquired inability to recognize words or language, although the speech frequencies, cadence and intensity variations of articulate speech were sensed. There was not sufficient audiometric loss in the speech frequencies to account for it. Sounds heard were useless as an aid to understanding speech. A hearing aid was of no use. In the opposite ear hearing was excellent. It is evident that threshold audiograms alone do not necessarily reveal the true capacity for understanding speech and are not infallible criteria for diagnosis or for prescribing of hearing aids.

A careful study of serial sections of the auditory tracts, nuclei and centers in subjects who have shown the phenomena I have described, and who have been properly examined clinically and audiometrically, will, I believe, aid in solving this mystery.

The following hypotheses are suggested as probable explanations . . . :

1. A psychological exclusion of the deafened ear to speech.

2. Insufficient speech pattern transmission or reception.

3. A lesion in the crossover pathways (to a contralateral dominant cortex). A lesion in the uncrossed pathways (to a homolateral dominant cortex).

4. An anoxemia depending upon circulatory stasis from autonomic nervous mechanism episodes, in the homo- or contralateral tracts, which temporarily or permanently eliminates one or both of these tracts sufficiently to interfere with reception of proper neural patterns by the dominant cortex.* (Too many pieces of the speech pattern are missing.)

[Corresponding to the asterisk, the author gives a footnote as follows: It has been shown that there is a splitting or rupturing of axons in the neuron at points of severe nerve trauma, whether toxic or mechanical, and that such lesions cause a dispersion or scattering of innervation to several localities by neurofibrils from a single axon.]

5. A total or crippling lack of crossing of the auditory pathways.

6. A total or unusual crossing of the auditory pathways. . . .

Analysis and correlation of the observations I have here set forth lead me to the conclusion that the syndrome described, i. e., monaural deafness for speech without sufficient loss of hearing for the speech frequencies to account for it, and without

15. Fowler, E. P.: Total Monaural Deafness for Speech, but Not for Speech Frequencies: Misleading Threshold Audiograms, Laryngscope **54**:605 (Nov.) 1944.

a loss of ability to remember or to produce speech properly and usually with a persistent tinnitus difficult to mask (and maybe a transitory vertigo) may in part at least be due to lesions central to the end-organ; in its primary nuclei or somewhere in the central auditory neurons to the same or the opposite side of the brain (or both), depending on whether the symptoms are in the ear homolateral or contralateral to the dominant cortex.

In a discussion of Dr. Fowler's paper Dr. Lorente de Nó states that the fact that the hearing frequencies are comparatively well heard shows that the cochlea is not too badly affected. The pathways from the cochlea and through the primary nuclei go to both sides of the brain. There are several crossings in the medulla, and the fibers from the two ears join at the level of the geniculate body, going together to the cortex. A lesion in the cortex or geniculate body would involve both ears, and therefore by exclusion one places the pathologic condition in the primary nuclei on the side where the hearing is defective so that the brain cannot analyze the incomplete constellation of impulses which it receives. Degeneration of fibers in the acoustic nerve and subsequent degeneration of neurons in the primary acoustic nuclei may produce the whole syndrome. Enough neurons are left to produce the sensation of hearing, but not enough to analyze the impulses. He believes a start should be made on histologic studies of acoustic centers of patients who have had proper clinical study. He knows of no complete description of the primary acoustic nuclei in man.

(Articles by Tunturi, Allen and Galambos, and Davis on cerebral localization of individual acoustic fibers, abstracted later in this review, are of interest in connection with the foregoing.)

Dr. L. Vosburgh Lyons calls this the Fowler phenomenon, as he knows of no previous description of it. Word deafness is a bilateral affair, because speech and sound memories are lost and incoming sounds heard are not correlated.

PHYSIOLOGY OF HEARING

Kellaway [16] in a recent article states that the perilymph is incompressible; hence movement of fluid within the cochlea can occur only by yielding of the round window. A low tone, 20 cycles, in a fluid such as the perilymph has a wavelength on the order of 60 meters, while the length of the fluid column between the oval and the round window is approximately 60 mm. The movement of the fluid is therefore a mass action. It follows, therefore, that immobilization at the round window should obliterate hearing. Culler, Finch and Girden cemented the round window and found the electrical effect and the acoustic reflex absent.

16. Kellaway, P.: Mechanism of the Cochlea, Arch. Otolaryng. **41**:252 (April) 1945.

Crowe and Hughson, using a cotton pledget or a periosteal graft over the round window, found cochlear potentials improved above 512 cycles.

Types of deafness referable to injuries of the nerve or center exhibit tone gaps in the upper range. It is impossible to explain these peculiarities without recourse to statistical analysis in the auditory end organ. Crowe, Guild and Polvogt, by histologic studies of ears giving audiograms with abrupt high tone losses, have verified this.

The author also analyzes experiments in stimulation deafness and the evidence from cochlear microphonics, effects of masking and surgical evidence.

The question of chemical mediator is gone into. Reference is also made to a study by Galambos and Davis, abstracted elsewhere in this review. The article should be studied in its entirety for full understanding.

Tunturi [17] in his article states that the cerebral cortex was explored for areas of reception of sound other than those reported previously. Restricted areas of the cochlea were stimulated electrically and the deflection read off from the various areas of the brain.

Destruction of ventral and dorsal areas did not affect the responses. Complete intracranial division of the eighth nerve abolished the responses in the three areas tested. After outlining the cortical area to shocks applied to the apical, middle and basal turns, the wires were placed 4 mm. lateral, medial and posterior to the basal turn. No response was obtained from this area of the cortex. Responses in this area were abolished by severing the modiolus, but reappeared on stimulating the central stump of the eighth nerve.

The cortical area described occupies the ventral end of the anterior ectosylvian gyrus in the cerebral cortex of the dog.

The use of masking and electrical stimulation of the cochlea furnished evidence of separate fiber connections from local parts of the cochlea to this area.

An article by W. F. Allen [18] states that complete bilateral destruction of the sylvian, ectosylvian and strips of the coronal and suprasylvian gyri prevented reappearance of correct conditioned differential response in one thousand one hundred tests for each dog and in additional tests six months later. Unilateral elimination of three areas permitted immediate return of the reflexes. Elimination of a part of these areas

17. Tunturi, A. R.: Further Afferent Connections to the Acoustic Cortex of the Dog, Am. J. Physiol. **144**:389 (Aug.) 1945.

18. Allen, W. F.: Effect of Destroying Three Localized Cerebral Cortical Areas for Sound on Correct Conditioned Differential Responses of the Dog's Foreleg, Am. J. Physiol. **144**:415 (Aug.) 1945.

disrupted, but permitted return of the responses after three hundred and twenty to four hundred and eighty-four tests.

Galambos and Davis [19] state that of 11 individual acoustic fibers studied for inhibition of spontaneous activity by one pure tone, 6 showed inhibitory areas, and 5 did not. All showed inhibition by two pure tones or by a rattle. It is not known why some fibers are inhibited by a limited range of the auditory spectrum while others are inhibited only by noises. The same results were noted but not so carefully studied in 25 other fibers.

There is considerable discussion of the nature of this inhibitory effect. The inhibitory effect of low tones on the reaction aroused by high tones is greater than that of high tones on low. The function of the inhibition is obscure, but it probably plays an important role in masking. The more effective masking tones in the cat have the same general distribution as those known to be particularly effective in human beings.

RESULTS OF HEARING TESTS

Gordon E. Peterson [20] gives the results of testing 3,393 children from the public schools of Indiana. The Western Electric 6B and Maico 5D audiometers were employed. One hundred children per day were checked, and a detailed description of the technic is given. Of the children 21.75 per cent failed the first test. Retests were given 571, and of these 8.66 per cent failed on second tests.

Jean Utley and Marion Goebel [21] give the results of testing 14 girls and 7 boys from the South Carolina School for the Deaf, aged 11 to 20, who were tested audiometrically with various hearing aids (not identified). One aid gave a gain of 14 per cent, and another 7 per cent. Some persons were helped by a loudspeaker system when the hearing aid failed. Several aids should be tried on any severely deaf child and tested with the pure tone audiometer.

Jean Utley [22] states that 51 pupils in a School for the Deaf, ranging from 9 to 24 years in age, 25 males and 26 females with various degrees of handicap, were tested. Forty-four were deaf before language was developed, 7 later. Freqencies of 128 to 2048 were tested on a Maico model D5 audiometer. Percentages of hearing loss were calculated by

19. Galambos, R., and Davis, H.: Inhibition of Activity in Single Auditory Nerve Fibers by Acoustic Stimulation, J. Neurophysiol. **7**:287 (Sept.) 1944.

20. Peterson, G. E.: The Pure Tone Screen Test of Hearing, J. Speech Disorders **9**:114 (June) 1944.

21. Utley, J., and Goebel, M.: A Comparative Analysis of the Results of Testing Individual Hearing-Aids in a School for the Deaf, Speech Monogr. **10**: 40, 1943.

22. Utley, J.: The Relation Between Speech Sound Discrimination and Percentage of Hearing Loss, J. Speech Disorders **9**:103 (June) 1944.

five methods, the methods of the American Medical Association, Fowler, Sabine, Western Electric Company and West. Consonants and vowels were tested separately and combined for discrimination ability and correlated with frequencies as well as percentage of hearing. The tests were presented over a group hearing aid to which the subjects were accustomed, and the ear phone was placed over the good ear. In applying the Fowler method for calculation of percentage of loss for speech, the recruitment factor was disregarded, as he recommended, in losses over 40 decibels.

The following conclusions were reached: Errors on both vowel and consonant tests showed a high correlation (0.84). Age and intelligence quotient seemed to be of little consequence and showed no significant relationship.

The correlation between losses at specific frequencies and sound discrimination increased with rise in frequency, being greatest at 1024 and 2048 for vowels and combined sounds, and 512, 1024 and 2048 for consonants.

When losses were estimated by any of the five methods for calculating percentage loss of speech, the correlations ranged from 0.85 to 0.88. This high correlation suggests that methods for computing percentage of loss may be more predictive of residual auditory discrimination than is often supposed. The absolute accuracy of different methods has yet to be determined.

Fordyce Johnson,[23] United States Army, reports a number of cases of deafness with undeveloped mastoid. He has made use of audiograms, and reports the findings of others. Seven references are given in the bibliography, which fails to mention the work of Witmaack on this subject.

TRAUMATIC DEAFNESS

Major C. M. Kos [24] states that traumatizing pressure changes occurring in the middle ear during ascent and descent cause only fleeting deafness when the eustachian tubes function adequately. When these pressure changes are not relieved on account of restricted permeability of the tube, tissue damage may result, made manifest by increased tension of the membrana tympani, interference with movement of the ossicles and exudate.

Fifty-four persons were studied, some by pretraumatic and posttraumatic audiograms. Ten with preexposure audiograms descended 38,000 feet (11,582 meters), 5 from 7,000 feet (2,133 meters), and 39

23. Johnson, F.: Deafness with Undeveloped Mastoid and Normal Tympanic Membrane, Arch. Otolaryng. **42**:174 (Sept.) 1945.

24. Kos, C. M.: Effect of Barometric Pressure Changes on Hearing, Arch. Otolaryng. **41**:322 (May) 1945.

had no preexposure audiograms. All stated that their trouble began at 5,000 feet (1,524 meters) or more. They were observed within a few minutes of injury and periodically until the hearing approximated normal. Forty sustained injury of both middle ears, and 14 unilateral. Representative audiograms illustrate the marked variations in hearing loss. The type of hearing loss usually considered typical for pressure deafness is depression of hearing for low tones with little if any loss for high tones. The author's graph supports the contention that the greatest number of ears incurred losses involving the higher frequency range and that the severest losses are found among the higher frequencies. Campbell believed this was due to superimposed auditory fatigue, but when the threshold markedly improves after inflation this contention is not supported. The deafness may not appear immediately after descent, but otoscopically the effusion may be watched accumulating in the middle ear.

The author advises, as primary treatment, nasal astringents, aspiration of excess secretions and frequent tubal inflations during the first few hours. In thousands of cases there have been no aggravations by this treatment. Those who showed only temporary improvement had high tone loss. Available data from several other sources indicate that only an exceptional few will sustain permanent disabilities purely from pressure trauma. Aero-otitis media is still a problem.

Major Lilburn C. Boemer [25] states that 310 patients with acoustic trauma came to otologic consideration, comprising 0.8 per cent of the patients admitted to the Twenty-First General Hospital from January 1943 to July 1944.

Twenty-five could hear low conversation at little over 3 feet (91.4 cm.), and 34 had average hearing of 8 feet (243.8 cm.) in either ear for low conversation. One hundred and eighty-one returned to general service duty with an average bilateral hearing of 4 feet (121.9 cm.) for low conversational voice.

The conglomerate audiogram showed mostly high tone loss, with initial depression at 2896 double vibrations per second. Maximum improvement takes place in two months or less, and more readily in younger persons.

Major Ullmann [26] states that 999 officers, including pilots, co-pilots, bombardiers and navigators, and 1,855 aerial gunners were tested for traumatic deafness. The majority had 600 to 2,500 flying hours. Among 100 officers, 66 had a fatigue notch at frequency 2896, 16 at

25. Boemer, L. C.: Acoustic Trauma, Ann. Otol., Rhin. & Laryng. **54**:513 (Sept.) 1945.

26. Ullmann, E. V.: Traumatic Deafness in Combat Flyers, Arch. Otolaryng. **40**:374 (Nov.) 1944.

4096, and 18 had mixed forms. The intensities of airplane noise have been measured and recorded as 80 decibels for soundproof planes and between 110 and 130 for open cockpit and combat planes. The various sources and types of noises were classified. It is noted that the four-engine Fortress increases the engine noise only a small fraction over a single engine plane. The most frequent dip was at 2896 double vibrations whether the exposure was to sudden blast or to continued noise. The ear is unprotected for five to ten milliseconds before the stapedius muscle and the tensor tympani contract. The damage was found by Crowe, Guild and Polvogt to be 2 or 3 mm. above the basal coil of the cochlea. This region is close to the round window. The author states that this area has a poor blood supply and therefore has decreased oxygen metabolism and greater susceptibility to fatigue. A definite relationship between hearing damage and hours of flying could be demonstrated.

In an article by Colonel Willard Machle [27] an initial survey of 45 gunnery instructors was used to establish the characteristics of the hearing loss in men frequently exposed to blasts of 37 and 75 mm. guns, and to an average of 100 rounds. Eighty per cent used improvised ear plugs of cotton, waste or rifle patches. Twenty per cent used them rarely or not at all. Repeated tests were made for four days prior to exposure. The better ear was the test ear; the control ear was protected by a fitted soft rubber plug, and the ear covered with a sponge rubber pad over which a cloth helmet held by a rubber band was fitted. Percentage loss of useful hearing was calculated by the method tentatively recommended by the Council on Physical Therapy of the American Medical Association.

A series of illustrative audiograms are shown. The author reports a high degree of acoustic protection with a number of devices. Significant loss was noted only when cotton or poorly fitted plugs were used.

The characteristics of the hearing loss were similar to those of deafness from exposure to sustained high noise levels. Acoustic injury is common in gunnery instructors, artillerists and others. With repeated exposure the damage is cumulative, but partial recovery of hearing occurs in the first few days after cessation of repeated daily exposures up to six or eight. After months of exposure there was partial recovery in only 2 of 10 men. There is a great variability in the response of individual subjects and uncontrollable variables in the behavior of blast waves, which prevents narrow definition of the resultant hearing loss.

27. Machle, W.: Effects of Gun Blasts on Hearing, Arch. Otolaryng. **42**:164 (Sept.) 1945.

In an article by Captain B. H. Senturia [28] 500 healthy men between the ages of 17 and 27 who had passed Class 1 requirements and had not been exposed to aircraft noise for thirty days were tested by audiometer for frequencies 1024 to 11584. (Lower frequencies were not tested because of suspected masking by ambient noise.) Hearing of plus or minus 10 decibels was classified as normal. Fairly uniform results were obtained by five examiners. Thirty-four per cent of right ears and 32 per cent of left ears were within 10 decibels. Tonal dips of 15 or more decibels occurred in 23 per cent of right and 29 per cent of left ears; 49.2 per cent showed bilateral similarity. Four types of audiograms were classified, and attention is called to the fact that significant deviation of these types may occur prior to exposure to occupational noise. It is suggested that this series of 500 audiograms be used as a control for field studies of postcombat cochlear damage.

Using a new type of audiometer measuring hearing for speech instead of pure tones, G. E. Shambaugh [29] found that patients who experience a greater than average gain from the fenestration operation have as good or better hearing than they formerly had from their hearing aid. Those with less than 20 decibels of improvement do not hear as well as they did with the hearing aid. On the basis of one year's results with the fenestration operation as now performed, ideal subjects have a 90 per cent chance for lasting improvements, and a 70 per cent chance that the hearing will equal or surpass that with a hearing aid.

By rehabilitation the author, M. M. Walter,[30] means any aids necessary to prepare the disabled person for employment, including medical diagnosis and treatment, and as applied to the deafened, a hearing aid, training in speech and speech reading, psychologic readjustment and education and training in a suitable occupation. He includes congenital deafness, deafness before language was developed and that acquired later. On the subject of hearing aids he gives the speech range as 256 to 2896 cycles. Best results are expected when the hearing loss averages between 40 and 70 decibels, but the author recommends hearing aids for losses of 20 to 45 decibels if the patients will wear them often enough to make them pay. For losses of 70 to 90 decibels he questions the practical benefit of hearing aids. Many other factors are discussed in detail. He ends by saying of vocational rehabilitation "that it conserves the greatest of all assets, the working usefulness of human beings."

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28. Senturia, B. H.: Auditory Acuity of Aviation Cadets, Ann. Otol., Rhin. & Laryng. **53**:705 (Dec.) 1944.

29. Shambaugh, G. E.: Comparison of the Improvement of Hearing Following the Fenestration Operation with That Obtained by Wearing a Hearing Aid, Arch. Otolaryng. **41**:189 (March) 1945.

30. Walter, M. M.: Vocational Rehabilitation for the Deaf and the Hard of Hearing, Arch. Otolaryng. **42**:157 (Sept.) 1945.

Drawing largely on experimental work done at Harvard over the years and on the known histopathology, M. H. Lurie [31] divides deafness into conduction or mechanical middle ear deafness and sense organ deafness, due to degeneration of the organ of Corti, and says that most deafness is due to a combination of these two types. Only in cases of acute infection of the middle ear is deafness due purely to infection. Deafness due to mumps is a true sense organ deafness.

Causes of the mechanical or conduction type are granulations, adhesions, etc., due to infection and fusion of the ossicles and of the stapes with the oval window. The relief by treatment is usually only temporary, as it is impossible to by-pass the fundamental cause. In otosclerosis the fenestra operation does by-pass the mechanical difficulties caused by the disease, and the author believes that its failure will be overcome by greater experience.

In changes in the organ or Corti due to loud sounds experimental evidence indicates that up to a certain point some of these are reversible; beyond this point the cells degenerate. In human beings there is a great variation in the reaction to loud sounds. Subjected to the same exposure one person makes a quick, another a slow recovery, and the author speculates as to whether or not there is a chemical mediator in the organ of Corti. The author decries the use of vitamins and endocrine substances in an attempt to cure this type of deafness. He purposely presents this pessimistic view because of the prospect of many cases of nerve deafness due to the war and because there will be much criticism that nothing is being done for them. Much will be done by psychologists, teachers of lip reading and by reeducation of the deaf generally, but not by otologists. Otologists cannot honestly offer any specific method as a cure for nerve deafness.

31. Lurie, M. H.: Deafness: Its Causes and What Can Be Done About It, Arch. Otolaryng. **42**:144 (Aug.) 1945.

Abstracts from Current Literature

Ear

TINNITUS, DEAFNESS AND DIZZINESS. J. KENT LEASURE, Laryngoscope **56**:54 (Feb.) 1946.

Tinnitus, deafness and dizziness can be of peripheral or of central origin. Whether it be either, the cause may be a local or a systemic disturbance. A careful history aids in the diagnosis. The peripheral lesion is more apt to have an abrupt onset with gradual improvement, while the central lesion starts gradually and usually becomes worse and worse. The examiner should inquire as to any systemic disease.

Some of the peripheral local lesions mentioned include wax, acute and chronic otitis media, obstruction of the eustachian tube and nasal disturbances. Infection about the orifices of the eustachian tubes is particularly important. Lesions of the internal ear can be detected by the use of the audiometer and the various tuning fork tests.

Peripheral systemic lesions may be caused by drugs, toxins of infections, glandular disturbances, allergy, neuritis of the eighth nerve and chronic degenerative processes.

Central lesions may be due to tumors, sclerosing or degenerative processes or injuries of the head.

HITSCHLER, Philadelphia.

PETROSITIS. RAUL VELASCO LETELIER, Rev. de otorrinolaring. **5**:256 (Dec.) 1945.

The author discusses 4 cases of petrositis. The most common organisms are hemolytic streptococci. The infection is usually transmitted either through the anterosuperior region of the promontory or through the peritubal cells. The clinical picture is usually that of a mastoidectomy which has been followed by persistent otorrhea and by otalgia that radiates to the orbital or the occipital regions. Diplopia is common, but the Gradenigo syndrome is not always diagnostic. The changes in the spinal fluid are not diagnostic, but the results of roentgen examination of the petrous tip are extremely important. Meningitis and septicemia are common complications. The condition must be distinguished from osteomyelitis of the petrous bone, thrombophlebitis of the lateral sinus, labyrinthitis and tuberculous meningitis. Chemotherapy is of little value preoperatively, but surgical treatment is of prime importance. The author uses the Lempert technic, but he believes that in any case in which the apex is not involved a complete thorough mastoidectomy is sufficient to clear up the symptoms of petrositis.

PERSKY, Philadelphia.

CEREBRAL ABSCESS. MARCUS COHN, Rev. de otorrinolaring. **5**:279 (Dec.) 1945.

Cohn presents 4 cases of cerebral abscess, in all of which the abscess was located in the temporoparietal lobe and followed an acute exacerbation of chronic suppurative otitis media. Meningitis was associated with the abscess in every case, and in 1 case paralysis of the third nerve appeared on one side and facial paralysis and hemiparesis of the opposite side. Extensive mastoidectomies were performed with exposure of the dura, and in 2 cases the result was death; in one death was due to leptomeningitis with rupture into the ventricle and in the other it was due to insufficient drainage and meningitis. Two patients recovered.

PERSKY, Philadelphia.

OTITIC MENINGITIS. J. DUCLOS, J. M. CORCUERA and O. IGUALT, Rev. de otorrinolaring. **5**:293 (Dec.) 1945.

The authors present 13 cases of otitic meningitis, in 7 of which the meningitis was what they term "sympathetic meningitis," evidenced by signs of meningeal irritation, increase in the spinal fluid pressure, increase in the cellular count up to 420 cells per cubic centimeter and a moderate increase in the serum albumin. However, this type does not show the presence of any particular organism, since it is purely a meningeal reaction to intense toxemia. In the other 6 cases the meningitis was of an acute purulent type, with the presence of pus and organisms. The authors attempted to distinguish between suppurative meningitis which followed acute mastoiditis and that which was associated with the chronic condition. In the former, the symptoms were always of shorter onset, and the temperature was higher and the toxicity much greater. In 4 of the cases of acute mastoiditis pneumococci were found; in 1 case, staphylococci, and in 1, streptococci. Staphylococci were found in every case in which meningitis was associated with chronic mastoiditis. Their operative findings in all cases revealed that the incidence of a pneumatic type of mastoiditis was greatest and that the incidence of a diploic type was second, while a sclerotic type occurred but rarely. At operations their most common finding was coalescent mastoiditis, but there was included among their cases osteitis of the middle portion of the petrous bone, a periphlebitic abscess, phlebitis, a cholesteatoma of the basal petrosa and finally a subdural abscess. Simple mastoidectomy was performed for acute mastoiditis, while radical mastoidectomy was done for chronic mastoiditis. Postoperatively in every case the patient received a sulfonamide drug, preferably sulfadiazine, supplemented with penicillin, which was given both intramuscularly and intraspinally. There was one death in this group of cases.

PERSKY, Philadelphia.

ACOUSTIC FUNCTIONAL TESTS AFTER BILATERAL FRACTURE OF THE PETROUS PORTION OF THE TEMPORAL BONE. PER PERMIN, Acta oto-laryng. **32**:40 (Jan.-Feb.) 1944.

Permin reports a case of skull fracture with bleeding from the left ear in which roentgen rays showed a fracture line beginning in the left parietal region, running downward and forward through the squama of the left temporal bone into the base and involving the region of the superior semicircular canal. There was no roentgenologic evidence of a fracture line in the petrous portion of the right temporal bone. An examination conducted five days later by another otologist revealed a pronounced tinnitus in both ears, no dizziness and no spontaneous nystagmus. There was clotted blood in the external canal of the left ear.

Seven days later the hearing had improved, there was no tinnitus, the right ear showed normal findings and there was a rotary-horizontal nystagmus to the left. During the following year there was a steady deterioration in the hearing of the left ear so that after twelve months the average threshold of perception for air-borne sound as measured by the audiometer was at 90 decibels. At the end of six months after the injury the acoustic function of the right ear was completely gone.

Permin calls attention to the findings of Skoog, who of 69 patients exhibiting longitudinal fractures of the temporal bone, found immediately after the injury a conductive deafness in 65 per cent, a perceptive deafness in 22 per cent and a combined conductive and perceptive deafness in 13 per cent. On the reexamination of these patients seven to thirteen years after the injury, he found normal hearing in 30 per cent, perceptive deafness in 54 per cent and the combined form in 16 per cent. Skoog also found that in the later series of examinations some of the patients showed a perceptive form of deafness who did not exhibit this form immediately after the injury.

The literature as examined by Permin indicates that the late diminution of hearing of a perceptive type following a longitudinal fracture may be due to a gradual periostitis ossificans in the sense of Ulrich, degenerative processes due to the absorption and organization of massive hemorrhages into the labyrinth, hemor-

rhages which have been demonstrated experimentally by Manasse, Stenger, Rhese and Voss, or by violent movements of the endolymph exerting a damaging effect on or causing destruction of the sensory epithelium as described by Wittmaack.

GROVE, Milwaukee.

AVIATOR'S DEAFNESS. RUTGER HEYDEN, Acta oto-laryng. **32**:164 (March-April) 1944.

This study is concerned with an investigation of the hearing function in the Swedish Air Force.

Theoretically the injuries to the ears of people flying in modern airplanes are of four kinds: (1) injuries due to large, rapid and frequent variations in barometric pressure, (2) injuries from intense noise during long periods of exposure, (3) injuries due to vibrations of relatively high pitch and (4) injuries due to long exposure to low atmospheric pressures.

The injuries caused by rapid climbing or rapid diving are usually located in the middle ear and are due to the inability of the pilot to equalize the pressure in his middle ear by voluntary opening of the eustachian tube. The aviator is unable to open his eustachian tube against a strong negative pressure because of underlying physical defects such as adenoids, septal deviations, hyperplastic rhinitis, chronic catarrhal tubotympanitis and acute inflammation in the nose and pharynx. These injuries to the middle ear are, to a large measure, preventable by proper prophylaxis.

The risk of acoustic damage becomes greater the larger the airplane engine used. The factors contributing to this damage are the noise of engine, the roaring of exhausts, the wind from the propeller and the vibrations of the plane. Furthermore, in order to be heard over the other masking noises the radio signals must be so loud that they also may cause real acoustic trauma.

The noise intensity to which an airplane crew is exposed varies from 80 decibels in a sound-isolated cabin to as much as 120 to 130 decibels in an open or plexiglass-covered cockpit.

Studies of the noise from airplane engines have shown that its components between 200 and 500 cycles are most intense and that the greater part of the noise in an airplane is below 1,000 cycles.

Heyden believes that the effects on the acoustic function of airplane vibrations and of prolonged exposure to low oxygen pressure are probably quite insignificant compared to damage from noise or from pressure changes.

Heyden found a perceptive type of deafness in about 30 per cent of his tested material and notes that this figure compares with 29.7 per cent as found in a 1940 examination of the Air Corps pilots of the United States Army.

GROVE, Milwaukee.

Larynx

ADENOMA OF THE BRONCHUS. H. J. MOERSCH, W. S. TINNEY and J. R. MCDONALD, Surg., Gynec. & Obst. **81**:551 (Nov.) 1945.

Thirty-eight consecutive cases of bronchial adenoma encountered at the Mayo Clinic are analyzed in this report. The authors conclude that the tumor is fairly common and point to its more frequent occurrence in relatively young persons. Females are more often affected than males. In the two latter respects the tumor contrasts markedly with bronchial carcinoma. Cough, hemoptysis, asthmatoid wheeze and recurrent episodes of pulmonary infection, in order of frequency, constitute the principal signs and symptoms. Although the bronchoscopic picture is fairly typical, final diagnosis must depend on microscopic examination. Vascularity of these tumors is a noteworthy feature. In the reported series there was no instance of metastasis to the opposite lung or outside the thoracic cavity. Treatment depends on the location of the tumor, the degree of secondary pulmonary

suppuration and the patient's general condition. Endoscopic destruction of the tumor or surgical removal by either lobectomy or pneumonectomy are proposed as the most satisfactory methods of treatment.

FRIEDBERG, Chicago.

ACQUIRED ESOPHAGOTRACHEOBRONCHIAL FISTULA. O. T. CLAGETT, J. H. PAYNE and H. J. MOERSCH, Surg., Gynec. & Obst. **82**:87 (Jan.) 1946.

The five main causes of esophagotracheobronchial fistulas are listed by the authors, in order of frequency, as (1) malignant disease of the esophagus, (2) infectious disease of the esophagus, trachea or pleura (tuberculosis, syphilis, fungus infections, suppurative esophagitis and nontuberculous empyema), (3) traumatic injuries of the esophagus, (4) esophageal diverticula and (5) esophagomalacia. Increasing dysphagia followed by coughing seizures while eating are the most prominent symptoms. The dangers of aspiration pneumonia, pulmonary abscess or gangrene are important considerations, particularly in fistulas not associated with malignancy.

The treatment varies with the cause. Some small fistulas of nonmalignant origin have been reported as successfully closed by chemical cautery; others, of larger size, have been surgically removed. The authors report a cure, by operative excision, of a fistulous communication between the esophagus and the right main bronchus. The pathologic report was "chronic inflammation and tuberculosis."

FRIEDBERG, Chicago.

TRACHEOTOMY IN LEPROSY. NORMAN R. SLOAN, Internat. J. Leprosy **1**:11, 1944.

Tracheotomy was done in 144 lepromatous patients over a period of twelve years. Forty-six are still living, and 98 have died.

Sloan feels that there is frequent involvement of the larynx in leprosy and that tracheotomy should be done early if life is to be prolonged. Most lepromatous patients have laryngeal stenosis if they live long enough.

A detailed description is given of the operative technic, the postoperative care, the surgical anatomy and the indication for the operation.

In general it is not good to wait until an emergency arises; when indications are present, the operation should be performed with careful attention to detail, especially control of bleeding.

"Chronic bronchitis" will eventually develop in tube-wearing patients, in time causing death of those who have not succumbed to intercurrent disease. This may be a few months after operation, or it may be more than seven years.

Five figures are included—three graphs and two operative sketches—and some frank and interesting reports of cases.

All in all tracheotomy is only a stopgap for some possible future chemotherapeutic agent which will really be effective in combating leprosy itself.

LIEBERMAN, Philadelphia.

Nose

SINUS DISEASE IN CHILDHOOD. GEORGE J. TAQUINO, New Orleans M. & S. J. **98**:8 (July) 1945.

The author emphasizes the facts that sinus disease does occur in young children and that the possibility of its occurrence is not generally recognized. The incidence is greater under poor economic conditions, although not extremely so. Boys are more often involved than girls because they are more careless in personal care. Its occurrence is more frequent in winter months, but the summer incidence is increasing because of the wider use of swimming pools. The defense mechanism of the sinuses depends on cell phagocytosis plus the protective blanket of mucus in the nose and the ciliary activity of the sinus mucosa. The nasal sinuses are usually well established by the third or fourth year. Ethmoid cells are present at birth and well developed at 2 years. Etiologic factors are colds, the exanthems, deviated

septums, enlarged adenoids, allergies and faulty hygiene. Allergies probably are factors in 50 per cent of all cases. Pathologically, sinus infections in children are classified as (1) purulent and allergic and (2) intumescent, catarrhal or purulent. The superimposed infection often dominant late in the disease sometimes obscures an allergic origin. The signs and symptoms are similar to those seen in adults—nasal discharge, cough, headache and otitis. Also, there are systemic signs and symptoms, such as anorexia, vomiting and failure to gain. The diagnosis of sinus infection in children requires much patience and tact. All the usual diagnostic procedures should be done, even if a light anesthesia is necessary.

Treatment.—Tonsillectomy is not to be performed until it is abundantly clear that it is necessary. It is suggested that removal should at least await the clearing of the infection, and then it may not be needed. Chemotherapy is to be avoided as a routine. Allergic sinusitis is not benefited by chemotherapy and must be managed as an allergy. Surgical procedures should be instituted when indicated. Radical operation is required only in extreme cases. Conservative measures are successful in 80 to 98 per cent of all cases.

BERKLEY, Los Angeles. [AM. J. DIS. CHILD.]

Miscellaneous

EFFECT OF GROWTH AND DEVELOPMENT ON ABNORMAL LABIAL FRENUM. ABRAHAM B. SCHWARTZ and THOMAS R. ABBOTT, Am. J. Dis. Child. **71**:248 (March) 1946.

A favorite saying of the old time family physician was that a child would "outgrow his deformity." This was not always true, but it was true enough to receive mention by such an astute observer as the late Dr. L. Emmett Holt (Growth as a Factor in Prognosis, *J. A. M. A.* **82**:1333 [April 26] 1924). Using this as a basis for their article, the authors state that they have followed a group of children with thickened labial frenum with associated separation of the upper central incisors. In a previous report photographs were shown of several such children. Recent photographs taken nine years later present a full complement of permanent teeth with no sign of abnormality. The thickened frenum had reverted to normal.

In dental literature a majority of oral surgeons (orthodontists in particular) seem to advise postponement of surgical intervention, at least until the permanent teeth have erupted; some still call for surgical excision when the frenum is abnormal. This gives rise to the question, when is it abnormal? The answer is that variations often tend to become normal if Nature has the time required to bring about this desired end. The brief article ends with the observation:

"During the intervening years since the first publication, we have seen no example of a thickened labial frenum which the natural process of growth and development did not remedy."

VOORHEES, New York.

SOME FURTHER EXPERIMENTS IN THE PRODUCTION OF NEGATIVE PRESSURE IN THE TRACHEA AND FRONTAL SINUS BY CILIARY ACTION. A. C. HILDING, Ann. Otol., Rhin. & Laryng. **54**:725 (Dec.) 1945.

Some time ago the author reported experiments which showed the production of negative pressure in the respiratory tract by ciliary action. It was found that if there was sufficient mucus present in the lumen of an air passage to block it, the ciliary action carried this along and produced negative pressure behind it, acting like a piston in a cylinder. It was suggested that this could explain post-operative atelectasis. It was demonstrated that absorption of air played no part in the process of developing negative pressure in the lumen of a hen's trachea by showing that a positive pressure was produced by reversing the position of the trachea with reference to the manometer.

The present work was done on a series of tracheas joined together with manometers inserted at the junctions. It was found that each additional trachea

added to the first showed an added increase in negative pressure. In the previous twenty-three experiments on tracheas, the highest negative pressure obtained was 55 mm. of water, and this was reached only in one. In these new experiments, 9 out of 20 tracheas mounted in series showed pressures above 55 mm., and 4 exceeded 100 mm. The highest recorded pressure was 146 mm.

The same phenomenon was also demonstrated in the frontal sinuses of dogs. With the animals under ether anesthesia, two 16 gage hypodermic needles were inserted in one frontal sinus through small holes made through the scalp and bone. Each needle was fitted so that it was airtight. One was connected with a manometer and the other to a syringe containing the mucinous secretion (collected from the tracheas of the dogs under experiment or from other dogs) to be injected into the sinus. A similar needle was inserted in the opposite sinus to act as a control and connected with another manometer. The mucus was injected, and negative pressure began to develop within the first minute or two. Negative pressure developed when the experiment was repeated after the dog was bled to death, and also when it was done after the dog was decapitated. When the animals were decapitated the possibility was removed of blood circulating through the sinus mucosa. The maximum negative pressure before decapitation was 38 mm. of water. After decapitation the experiment on the same head again developed negative pressure, but only up to 22 mm. The experiment was repeated on the sinus of 1 dog which had been used as control, and a greater negative pressure was obtained than in the other side either before or after decapitation. Since the negative pressure can be produced as readily immediately after death as before, one must conclude that it is not due to absorption of air by the blood stream.

MILLER, Philadelphia.

TONSILLECTOMY AND POLIOMYELITIS. C. E. IRWIN, Arch. Surg. **49**:136 (Aug.) 1944.

The author reviews several papers that discuss the relationship of tonsillectomy to poliomyelitis. Reference is made to McCormick's study that presents a new concept of the etiologic relationship of lymphatic hypertrophy to degenerative diseases of the central nervous system. In pointing out the higher incidence of poliomyelitis in tonsillectomized children, the author raises the question of the possible etiologic relationship of the basic constitutional condition of the patient which is responsible for the lymphatic involvement necessitating the tonsillectomy and the effect of the anesthesia from the standpoint of nutritional deficiency and anoxia.

KAISER, Rochester, N. Y. [AM. J. DIS. CHILD.]

PENICILLIN IN THE TREATMENT OF PNEUMOCOCCIC MENINGITIS: A STUDY OF 67 CONSECUTIVE CASES. EMANUEL APPELBAUM and JACK NELSON, J. A. M. A. **128**:778 (July 14) 1945.

Sixty-seven patients with pneumococcic meningitis were treated with penicillin administered intrathecally, with total doses varying from 10,000 units to 700,000 units. In a few cases intracisternal, intraventricular or subdural administration was employed. All but 3 of the patients received additional penicillin intramuscularly or intravenously. Treatment with sulfonamide compounds, usually sulfadiazine, was given prior to or in combination with the penicillin treatment in almost all cases. The mortality rate was 61 per cent. Factors adding to the difficulty of successful treatment are the presence of primary foci of infection, the presence of bacteremia and the occurrence of the disease in fulminating form.

BAIRD, Wauwatosa, Wis. [AM. J. DIS. CHILD.]

STUDIES OF FIBRIN FOAM AS A HEMOSTATIC AGENT IN NEUROSURGERY, WITH SPECIAL REFERENCE TO ITS COMPARISON WITH MUSCLE. FRANC D. INGRAHAM, ORVILLE T. BAILEY and FRANK E. NULSEN, J. Neurosurg. **1**:171 (May) 1944.

A completely satisfactory hemostatic material for neurosurgical work should produce prompt control of bleeding, should give no (or very slight) tissue reaction

and should not cause the formation of antibodies. The authors report the results of a number of experiments showing the superiority of fibrin foam and thrombin to other agents.

The escape of a large amount of thrombin into the cerebrospinal fluid is not followed by any serious reactions. As compared with muscle thromboplastin, soaked fibrin foam appeared to have the following advantages: 1. It produces far less tissue reaction. 2. The supply is virtually unlimited. 3. An excess is innocuous. 4. It can be molded to conform to the contour of the bleeding surface. 5. If dislodged free in the ventricle, it can be absorbed. 6. Its use is superior in large bleeding channels, where a large plaque is required. 7. It is usable where muscle could seldom be used, thus saving much time at operation.

There is a considerably greater tissue reaction to black silk and bone wax than to fibrin foam. Furthermore, fibrin foam is better than soluble cellulose in that (1) the foam structure holds the thrombin better than the fibrillar cellulose; (2) the fibrin foam is more readily cut into adaptable shapes and will better retain the contours of the bleeding surface; (3) it is more quickly and permanently adherent to the bleeding surface.

WHITELEY, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

ADENOCARCINOMA, CYLINDROMA TYPE, OF THE PAROTID GLAND. F. W. QUATTLEBAUM, M. B. DOCKERTY and C. W. MAYO, Surg., Gynec. & Obst. **82**:342 (March) 1946.

In a clinical and pathologic study of 210 consecutive primary parotid neoplasms at the Mayo Clinic, the authors found an incidence of 10 per cent, or 21 cases, of adenocarcinoma of the cylindroma type. They believe that there is no justification for inclusion of this particular group in the category of mixed salivary tumors. Sharp pain and local fixation of the growth were often present, and surgically the growths were generally more infiltrative than encapsulated. In these respects they differ markedly from mixed tumors. In the entire series only 5 patients, or 25 per cent, are known to be living and well without recurrence. This low percentage suggests that solid tumors of the parotid gland should be assumed to be malignant until proved otherwise, and that more radical surgical procedures are apparently indicated in order to better the end results.

FRIEDBERG, Chicago.

TUBERCULOSIS OF THE ESOPHAGUS. C. A. HAMBERGER, Acta oto-laryng. **33**:281 (July-Aug.) 1945.

Hamberger calls attention to the rarity of tuberculosis of the esophagus. Up to 1926 only 116 cases had been reported. He feels that it is astonishing that the esophagus is not more frequently involved when one considers that it is continuously bathed by saliva containing the tubercle bacillus. He thinks that this immunity to infection of the esophagus is due to the pavement character of its epithelium.

There are five ways in which tuberculosis of the esophagus may develop: 1. The mucosa may be infected during the act of swallowing infected saliva. This mode of infection is impossible if the mucosa is intact but could readily occur after a stricture. 2. In connection with tuberculosis of the larynx the invasion of the esophagus could occur by a direct propagation. 3. Caseous tuberculous peribronchial or peritracheal glands can penetrate the wall of the esophagus. 4. Infection may start by way of the blood stream. 5. There may be a lymphatic propagation in cases in which there are tuberculous glands or a tuberculous pleurisy. The upper third of the esophagus is the most frequently involved, the inferior third the least frequently. Tuberculosis of the esophagus presents itself in ulcerative and sclerotic stages. The sclerotic type leads to progressive stenosis. The ulcerative form produces painful deglutition. The best diagnostic results are obtained by endoscopy. The prognosis depends on the general condition of the patient. The author describes a case in which the condition was diagnosed by endoscopic methods.

GROVE, Milwaukee.

News and Comment

APPEARANCE OF A NEW JOURNAL FOR PLASTIC SURGERY

For the rhinoplastic surgeon the most significant news of the moment concerns the appearance of the first number (July 1946) of the magazine *Plastic and Reconstructive Surgery*. Although the magazine is published by the American Society for Plastic and Reconstructive Surgery, Inc., suitable papers and reports of original work will be accepted from all fields of plastic surgery, and the contributors are not to be limited by membership in the society. This adds to the scope and the value of the publication, as will the abstracts of the current literature on the subject. The stimulation of contributions of new and original theories and methods will also add to the worth of the magazine. It will be recalled that when the Rockefeller Institute for Medical Research was dedicated a visitor of prominence remarked to the director that if he did not allow his assistants to do work that at the time seemed foolish they would make no great discoveries. (This was Dohrn's remark to Dr. Simon Flexner, reported in the latter's biography of Dr. William Welch.) In support of this idea, which may well be applied also to papers submitted to editors for publication, one has only to consider the struggles of such students as Pasteur and the Curies for recognition of their pioneer work.

The notable and representative names on the editorial board of the magazine give promise of adequate fulfilment of anticipation.

In this first number of the magazine a historical presentation by Aufricht gives a clear picture of the work done by men in the United States in developing the art of plastic surgery, as well as of the more familiar very early beginnings. The bibliographic list adds to the interest of the article. J. S. Davis contributes detailed consideration of the use and the value of the Z plastic operation as used in the correction of congenital and other deformities. He presents a clear picture of the method. The use of Z flaps in cases of circular constriction is demonstrated by Stevenson. Sutton describes means and methods for restoring function of the arm when there is contraction of the tissues of the axilla. Extensive traumatic deformities of hands and feet for which early covering is required are dealt with by McDonald and Webster. The subject of coverage and of use of suitable grafts in the repair of genital and urethral injuries is carefully detailed by Robinson, Stephenson and Padgett. Jaeger presents a report on the results of the use of various materials for closing skull defects that have resulted from injuries. Other papers deal with surgical methods of dealing with cutaneous lesions ("Icthyosis Hystrix" by Kazanjian); a new method of managing split skin grafts with Dermatape (Reese); the therapeutic tattooing of capillary hemangiomas with white pigment (Brown, Cannon and McDowell); an experimental study of the growth of young human cartilage grafts (Peer); the use of motion pictures, lantern slides, photographs and models to advance the teaching of the operative technics of plastic surgery (Palmer).

None of these papers are directly applicable in the field of rhinoplasty, though any method used for plastic repair may be adaptable to reconstructive operations on or about the nose or the head. The only exception is a paper by Straith on the elongation of the columella.

PANEL DISCUSSION IN DETROIT—ASSOCIATION OF MILITARY SURGEONS

The Association of Military Surgeons will hold an Eye, Ear, Nose and Throat Panel Discussion on Friday, Oct. 11, 1946, in the English Room of the Statler Hotel, Detroit.

Major General Norman T. Kirk, M. C., U. S. A., and Vice Admiral Ross T. McIntyre, M. C., U. S. N., will be the honorary chairmen.

The program will be as follows:

9:00 A. M.

"Traumatic Injuries of the Eyes and Their Repair." Commander A. Duane Beam, M. C., U. S. N. R., Detroit.

"Rehabilitation of the Blind." Lieutenant Colonel James N. Greear, M. C., A. U. S., Washington, D. C.

"Repair of Eye Injuries: A Motion Picture." Lieutenant Colonel Gilbert C. Struble, M. C., A. U. S., Valley Forge General Hospital, Phoenixville, Pa.

"X-Ray Reconstruction of Orbit as an Aid to Surgical Reconstruction." Lieutenant Colonel E. L. Shiflett, M. C., A. U. S., William Beaumont General Hospital, El Paso, Texas.

"Ear Plugs for the Prevention of Deafness." Lieutenant Colonel Ben. H. Senturia, M. C., A. U. S., St. Louis.

"Irradiation of the Eustachian Tubes." Lieutenant Colonel Edmund P. Fowler Jr., M. C., A. U. S., New York.

2:00 P. M.

"Laryngeal Injuries and Their Repair." Lieutenant Colonel Mercer G. Lynch, M. C., A. U. S., New Orleans.

"Rehabilitation of the Deaf in the Navy." Captain Francis L. Lederer, M. C., U. S. N. R., Chicago.

"Facial Nerve Repair Due to War Injury." Lieutenant Colonel Frank D. Lathrop, M. C., A. U. S., Boston.

"Lempert Fenestra Nov-ovalis Operation for the Restoration of Practical Serviceable Hearing in Clinical Otosclerosis: A Motion Picture of the Technic." Dr. Julius Lempert, New York (by invitation).

NEW MEMBERS OF THE AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

At the Twenty-Seventh Annual Meeting of the American Broncho-Esophagological Association, held May 31, 1946, in Chicago, the following applicants were elected to membership:

Active Membership

Dr. Charles D. Blassingame, Memphis, Tenn.
Dr. Lawrence R. Boies, Minneapolis
Dr. Glenn J. Greenwood, Los Angeles
Dr. Elmer W. Hagens, Chicago
Dr. Archibald R. Judd, Hamburg, Pa.
Dr. Paul L. Mahoney, Little Rock, Ark.
Dr. Julius W. McCall, Cleveland
Dr. Paul W. Moore Jr., Cleveland
Dr. Ralph G. Rigby, Salt Lake City
Dr. Charles C. Wolcott, Yonkers, N. Y.
Dr. Sterling C. Yinger, Springfield, Ohio

Corresponding Membership

Dr. Jose Ameriso, Rosario, Argentina
Dr. Ermiro Estevam de Lima, Rio de Janeiro, Brazil
Dr. Manuel Gonzales Loza, Rosario, Argentina
Dr. Anibal Grez, Santiago, Chile
Dr. Guido Guida, Rome, Italy
Dr. Abelardo Irigoyen Freyre, Santa Fe, Argentina
Dr. Jose Arthur de Carvalho Kos, Rio de Janeiro, Brazil
Dr. Roger Lanza Castelli, Cordoba, Argentina
Dr. Ernesto Raffo, Lima, Peru
Dr. Juan Manuel Tato, Buenos Aires, Argentina

Honorary Membership

Prof. Felix R. Nager, Zurich, Switzerland

Dr. Robert M. Lukens, Philadelphia, is president, and Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago, secretary.

Directory of Otolaryngologic Societies *

INTERNATIONAL

First Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.
Place: Palmer House, Chicago. Time: Oct. 17-19, 1946.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles.
Secretary: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Place: Atlantic City. Time: June 9-13, 1947.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
President-Elect: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 13-18, 1946.

American Broncho-Esophagological Association

President: Dr. Robert M. Lukens, Medical Arts Bldg., Philadelphia 2.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11.
Place: St. Louis.

American Laryngological Association

President: Dr. Frank R. Spencer, Physicians Bldg., Boulder, Colo.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.
Place: St. Louis. Time: April 23-24, 1947.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Harry W. Lyman, 906 Carleton Bldg., St. Louis.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: St. Louis. Time: April 25-26, 1947.

SECTIONS:

Eastern.—Chairman: Dr. Robert F. Ridpath, 18th and Chestnut Sts., Philadelphia.
Place: Philadelphia. Time: Jan. 13, 1947.
Southern.—Chairman: Dr. Walter T. Hotchkiss, 541 Lincoln Rd., Miami Beach, Fla.
Place: Miami, Fla. Time: Jan. 6, 1947.
Middle.—Chairman: Dr. Oliver E. Van Alyea, 135 S. La Salle St., Chicago.
Place: Chicago. Time: Jan. 20, 1947.
Western.—Chairman: Dr. Colby Hall, 1136 W. 6th St., Los Angeles, Calif.
Place: Los Angeles. Time: Jan. 25-26, 1947.

American Otological Society

President: Dr. William E. Grove, 324 E. Wisconsin Ave., Milwaukee, Wis.
Secretary: Dr. Gordon D. Hoople, 713 E. Genesee St., Syracuse, N. Y.
Place: St. Louis. Time: April 21-22, 1947.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Alfred Schattner, 115 E. 61st St., New York.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven, Conn.
Place: New York. Time: Nov. 14, 1946.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.